A Dissertation on

"A STUDY ON THE SERUM BETA HCG AND LIPID PROFILE IN EARLY SECOND TRIMESTER AS PREDICTORS OF PREGNANCY INDUCED HYPERTENSION"

Dissertation Submitted to

THE TAMIL NADU Dr.M.G.R. MEDICAL UNIVERSITY

CHENNAI- 600032

with partial fulfillment of the regulations

for the award of the degree of

M.S.OBSTETRICS AND GYNAECOLOGY

(BRANCH 1)

COIMBATORE MEDICAL COLLEGE,

COIMBATORE

MAY 2018

CERTIFICATE

Certified that this is the Bonafide Dissertation in **"A STUDY ON THE SERUM BETA HCG AND LIPID PROFILE IN EARLY SECOND TRIMESTER AS PREDICTORS OF PREGNANCY INDUCED HYPERTENSION"** was a work done by **Dr.NITHYA.S**and submitted in partial fulfillment of the requirements for the Degree of **M.S.Obstetrics and Gynaceology**of The Tamil naduDr.M.G.R Medical University, Chennai.

Signature of the Guide

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DECLARATION

I Solemnly declare that the Dissertation titled "**A STUDY ON THE SERUM BETA HCG AND LIPID PROFILE IN EARLY SECOND TRIMESTER AS PREDICTORS OF PREGNANCY INDUCED HYPERTENSION"** was done by me at Coimbatore Medical College during the academic year July 2016 – June 2017 under the guidance of **Prof. Dr.Murugalakshmi M.D., DGO.,**this Dissertation is submitted to the TamilnaduDr.M.G.R Medical University towards the fulfillment of the requirement for the award of **M.S. Degree in Obstetrics and Gynaecology**

PLACE: **Dr. NITHYA. S**

DATE:

ACKNOWLEDGEMENT

It's my privilage to express my sincere thanks to**Dr.B.Asokan, M.S,MCh,the Dean,**Coimbatore Medical College Hospital for permitting me to utilize the clinical materials of this hospital.

It gives me immense pleasure to express my deep sense of gratitude and sincere thanks to my guide **Dr.Murugalakshmi,M.D.,D.G.O.,** for her guidance,suggestion,advice and constant encouragement during the course of my study.

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I thank my colleagues,CRRI's and staff nurses who have been a source of constant help.

I am indebted to my patients who have submitted themselves to this study.

I am grateful to my parents who are a constant source of inspiration and support.

CERTIFICATE – II

This is to certify that this dissertation work titled "**A STUDY ON THE SERUM BETA HCG AND LIPID PROFILE IN EARLY SECOND TRIMESTER AS PREDICTORS OF PREGNANCY INDUCED HYPERTENSION"** of the candidate **DR.NITHYA.S**with registration Number **221516302** for the award of **M.S in the branch of M.S. Degree in Obstetrics and Gynaecology**, I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains 78 pages from introduction to conclusion and the result shows **5% (Zero)** percentage of plagiarism in the dissertation.

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Course : POST GRADUATE IN MS OBSTETRICS AND GYNAECOLOGY

Period of Study : ONE YEAR

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Dissertation Topic : A STUDY ON THE SERUM BETA HCG AND LIPID PROFILE IN EARLY SECOND TRIMESTER AS PREDICTORS OF PREGNANGY INDUCED HYPERTENSION

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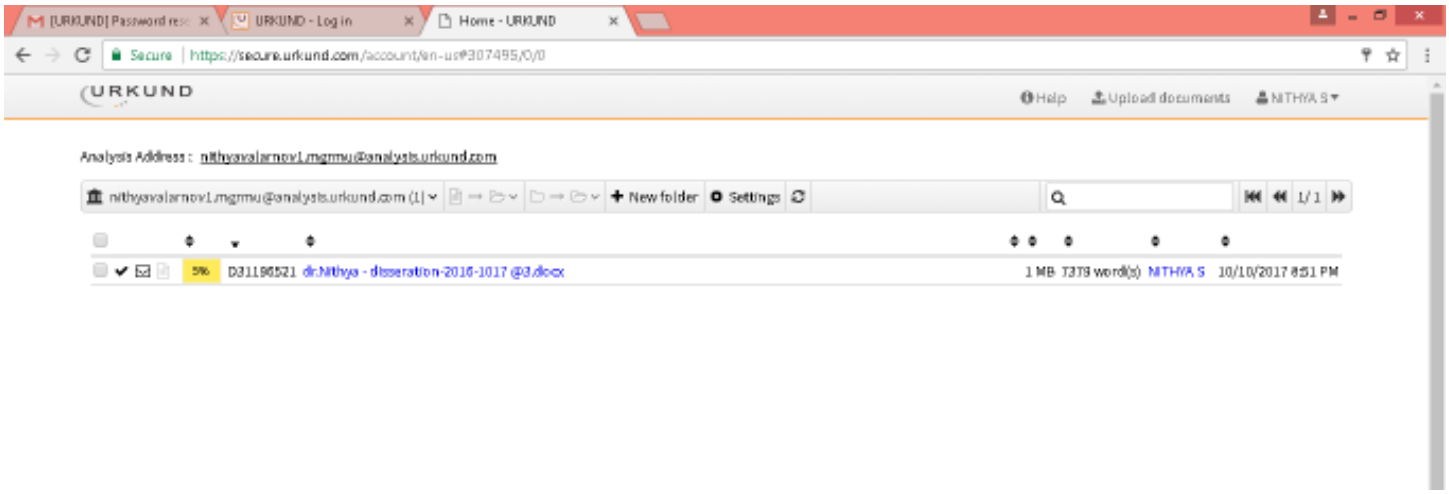
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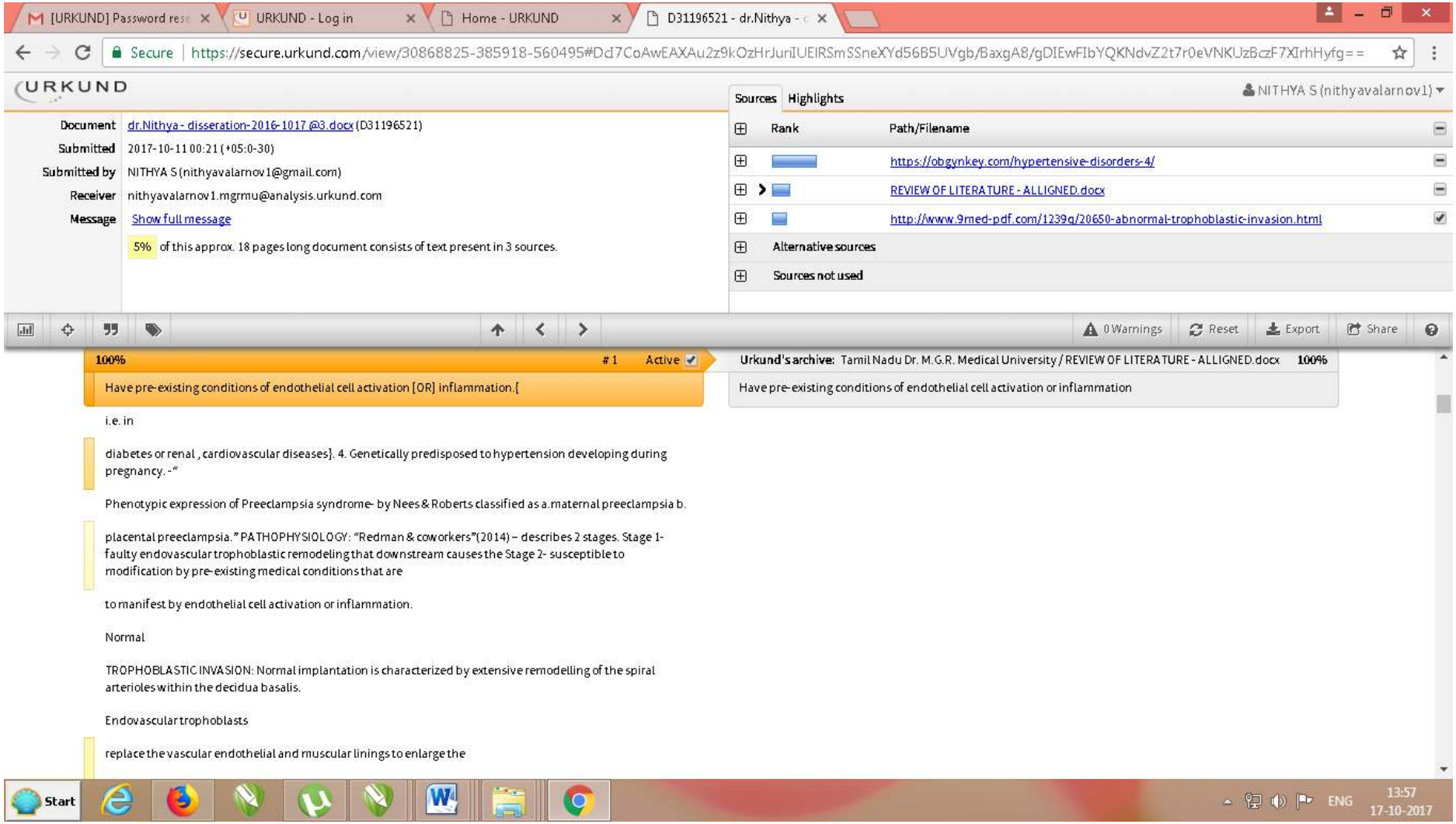
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Normal
TROPHOBLASTIC INVASION: Normal implantation is characterized by extensive remodelling of the spiral arterioles within the decidua basalis.
Endovascular trophoblasts
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https://obgynkey.com/hypertensive-disorders-4/
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Instances where selected sources appear:

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INTRODUCTION

Pregnancy is nature's precious gift utilizing it's 9 months of duration in achieving good maternal and fetal outcome. There are several complications that may impair a favourable pregnancy outcome. Preg

nancy Induced Hypertension is one of the most common medical disorder encountered next to Anemia.*Cunningham et al* described PIH-" major cause of MATERNAL morbidity and mortality".

Hypertension complicates 5 to 10% of all pregnancies worldwide. Pregnancy induced Hypertension accounts for 3.9% of all pregnancies.

Yagnik et al and Susane proposed that Uncontrolled pregnancy induced hypertension can lead to maternal and fetal complications and increased risk of hypertension and diabetes in the fetus,in the later stages of life.Hence the above study was done in which both serum Beta- hCG and serum lipid profile was used as screening tests to predict preeclampsia in the early second trimester.

AIM OF THE STUDY

The study aims at testing the hypothesis that women with high serum beta hcg levels and lipid profile in early second trimester have high risk of developing pregnancy induced hypertension.

OBJECTIVES OF THE STUDY

This study is undertaken mainly to forecast the scenario of developing pregnancy induced hypertension at early trimester of 14 to 20 weeks of gestation by two main predictors – serum beta hcg and plasma lipid profile to prevent the development of eclampsia at near term.

b-hcg estimation was done by enzyme linked immunosorbent assay method and serum lipid profile by enzymatic calorimetric test with lipid clearing factor.

METHODOLOGY

SOURCE OF DATA:

A total of 150 pregnant women who attended the antenatal clinic of the department of obstetrics and gynaecology at Coimbatore medical college hospital as well as those admitted in the ward were selected for the study.

STUDY PERIOD:

MAY 2016 TO SEPTEMBER 2017.

STUDY DESIGN:

Prospective study.

STUDY SUBJECTS:

Sample size:150

INCLUSION CRITERIA:

-those with known last menstrual period or first trimester usg screening with gestational age between 14 to 20 weeks irrespective of parity.

-detailed history- age, parity, height,pre-pregnancy weight and weight at the time of collection of blood sample.

-family history of hypertension, physical activity during pregnancy were noted.

-patients with edema,high blood pressure were included.

EXCLUSION CRITERIA:

-Women with hypertension diagnosed before 20 weeks of gestation.

-Diabetes Mellitus.

-Multiple pregnancy.

-Ultrasound proved CONGENITAL MALFORMATIONS

-Molar Pregnancy.

REVIEW OF LITERATURE

EPIDEMIOLOGY:

A systemic review by World Health Organisation reveals that "hypertensive disorders account for 16% of all deaths in the developed countries", with

9% maternal deaths in Africa and Asia,

26% maternal deaths in Latin America and Carribean.

INCIDENCE:

It depends upon

-Race

-Ethinicty

-Parity

-Genetic predisposition

In the United states, hypertensive disorders in pregnancy alone represents 5 to 8% of all livebirths.

Incidence rates in various countries:

According to study by the *"Maternal and Fetal Medicine Unit Network"[*MFMN] conducted in 2400 nulliparous women,the percentage of preeclampsia is as follows:

5% - WHITES

9% - HISPANICS.

11% - AFRICAN-AMERICAN.

Stafford and coworkers in 2004 made a study in which Nulliparous > Multiparous are at high risk of preeclampsia'.

This study was made among people from various countries ,namely. Australia, Canada, Denmark, Norway, Scotland, Sweden and Masachusettes.

In accordance with data from the United National Hospital Discharge Survey, the rate of preeclampsia during admission increased by 25% from 1987 to 2004.

Severe morbidity associated with preeclampsia and eclampsia include renal failure, stroke, cardiac dysfunction or arrest,respiratory compromise, coagulopathy and liver failure.

RISK FACTORS:

These are classified into 2 types

Based on - Pregancy specific charactersitics

- Maternal pre-existing features., that lead to rise in preeclampsia.

Pregancy specific charecteristics: 1. Parity

2. Placental factors

3. Age.

4. Race.

PARITY:

Based on systematic review of controlled studies, nulliparity triples the risk of preeclampsia.

Two-thirds of pih in primi progress beyond first trimester. Pathogenesis behind this is the Immunological phenomenon .

Hence, previous H/o pregnancy loss, increased duration of sexual activity prior to pregnancy, prolonged pre-pregnancy cohabitation – decreased PIH.

PIH increases with - use of barrier Contraceptives

new paternity,Donor Sperm Insemination.

PLACENTAL FACTORS :

Excess placental volume leads to high incidence of pih as in multifetal gestation and Hydatidiform mole.

AGE:

Extremes of childbearing age have higher incidence of preeclampsia, i.e,bipolar distribution.

Women aged 40 yrs and nulliparous are at risk

Risk ratio - 1.68

Primparous - 1.23 to 2.29

Multiparous - 1.34 to 2.87

Apart from the above risk factors, the following also affect the occurrence of the PIH.

1. Social, Environmental and Seasonal changes also increase the risk of PIH [*Lowler ,2005] &[Palmer,1999]*.

2.BMI- the relationship between the WEIGHT and risk of preeclampsia is progressive.

i.e if BMI- 20kg/m2- risk is 4.3%

BMI- 35kg/m2- risk is 13.3%

3.Hyperhomocystinemia.

4.Obesity- Metabolic syndrome.

5. A 30% increase in PIH is seen in EXTRAUTERINE

GESTATION > 18 weeks of gestation.

6.Race- More severe froms of preeclampsia are associated with Maternal NON-WHITE race

7.Pre-existing diseases-

Chronic hypertension – 10-25% risk

Pre-gestational diabetes - 21% risk.

Long standing Diabetes associated

with microvascular disease - 36 to 54% risk.

With mild renal disease[Cr<1.5] - 20 to 25%

With severe renal disease - >50% risk.

Significant high risk with Autoimmune diseases

Namely SLE, APLA[Antiphospholipid Antibody Syndrome].

-Paradoxically, SMOKING decreases the risk of Preeclampsia due to the increased placental volume homeostasis by adrenomodullin expression and modulation of the angiogenic factors.

AETIOPATHOGENESIS:

AETIOLOGY:

Gestational hypertension develops when the below criteria are met:

1.There is exposure to chorionic villi for the first time.

2. There is exposure to superabundance of chorionic villi for the first time i.e as in hydatidiform mole and twins.

3. Have pre-existing conditions of endothelial cell activation [OR] inflammation.{i.e. in diabetes or renal , cardiovascular diseases}.

4. Genetically predisposed to hypertension developing during pregnancy.

-" Phenotypic expression of Preeclampsia syndrome- by *Nees & Roberts* classified as

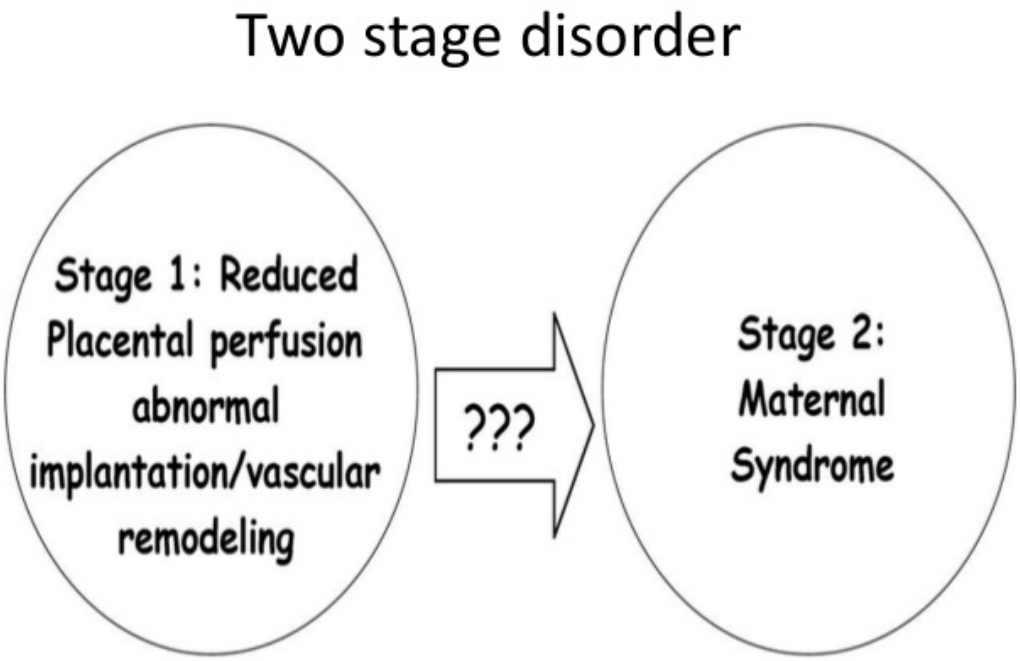
a.maternal preeclampsia b. placental preeclampsia."

PATHOPHYSIOLOGY:

"Redman & coworkers"(2014) – describes 2 stages.

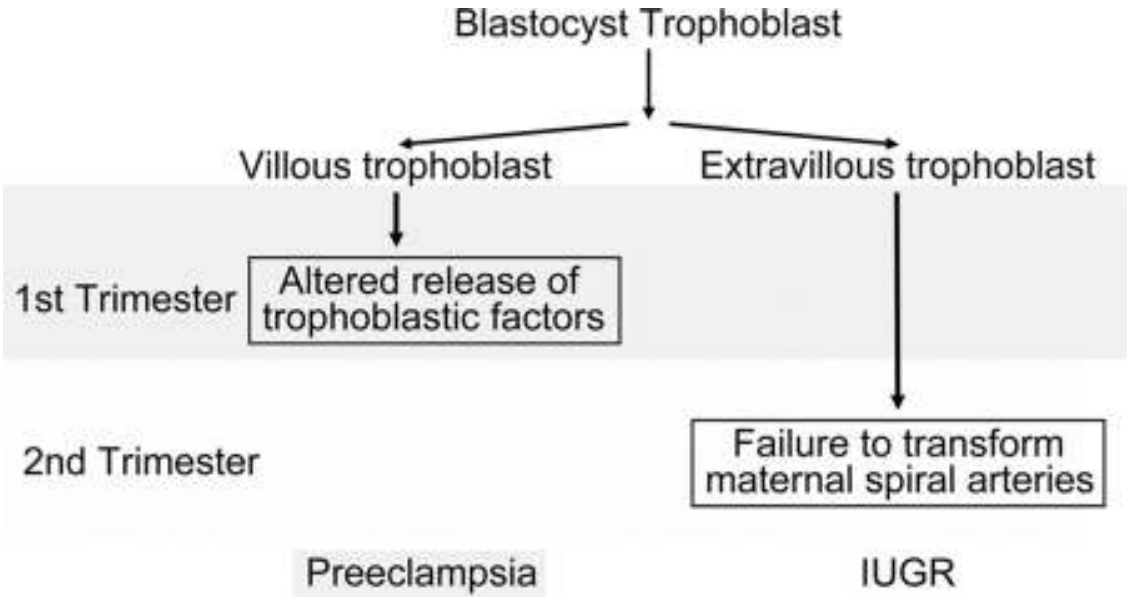
Stage 1- faulty endovascular trophoblastic remodeling that downstream causes the

Stage 2- susceptible to modification by pre-existing medical conditions that are to manifest by endothelial cell activation or inflammation.



Normal TROPHOBLASTIC INVASION:

Normal implantation is characterized by extensive remodelling of the spiral arterioles within the decidua basalis. Endovascular trophoblasts replace the vascular endothelial and muscular linings to enlarge the vessel diameter. The veins are invaded only superficially.



ABNORMAL INVASION:

In Preeclampsia, there is INCOMPLETE TROBPHOBLASTIC INVASION- secondary wave.

With this, only the decidual vessels and not the myometrial vessels becomes lined with the endovascular trophoblasts – 'the deeper myometrial arterioles do not lose their endothelial lining and musculoelastic tissue and their mean external diameter is only half that of the corresponding vessels in the normal placentas."

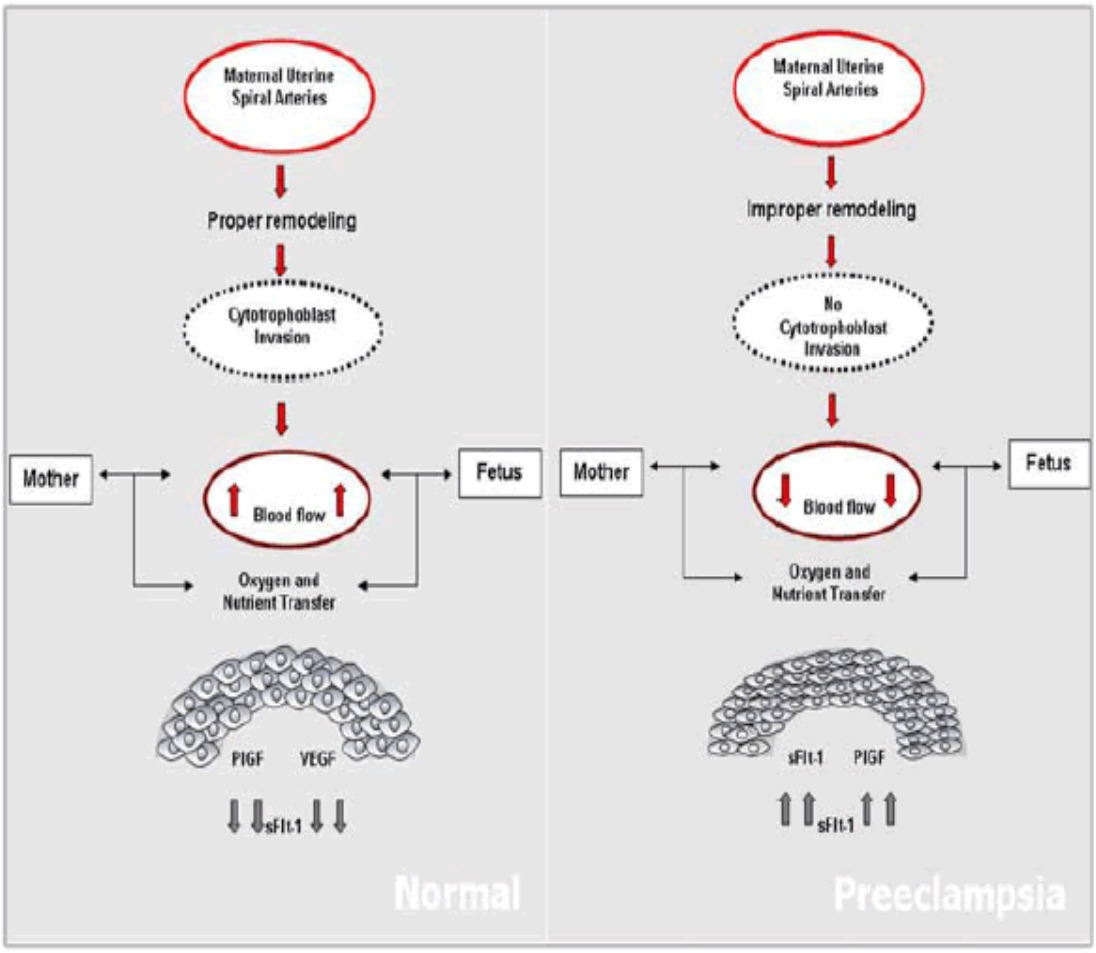
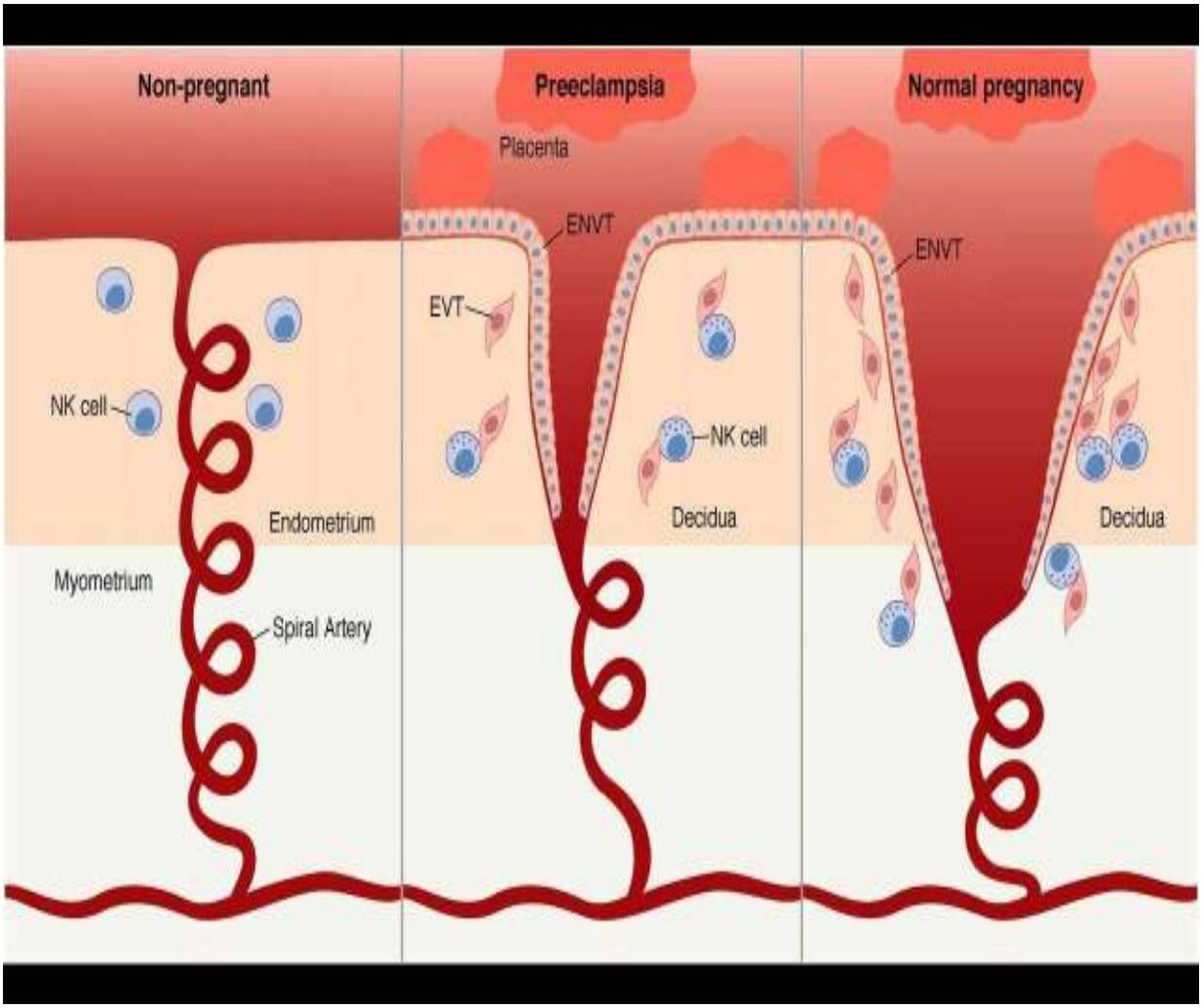


Figure 1. Endothelial dysfunction in preeclampsia

The left panel shows condition in normal subjects while the right panel shows situation in subjects with preeclampsia.



The most common mechanisms which lead to the causation of preeclampsia are:

1. VASOSPASM

2. ENDOTHELIAL CELL INJURY.

3. IMMUNOLOGICAL FACTORS.

4. PLACENTAL PATHOLOGY.

VASOSPASM:

This concept leading to preeclampsia was formulated by *VOLHARD {1918}*.

"Endothelial cell damage ⟶ interstitial leakage ⟶ causes the blood constituents{platelets, fibrinogen}to be deposited subendothelially ⟶ disruption of the endothelial junctional proteins ⟶ ultrastructural changes in the subendothelial region of the resistance arterioles in preeclamptic women".

In normal pregnant women, there is increased refractoriness to infused vasopressors. In preeclampsia, increased sensitivity to angiotensin II is present.

ENDOTHELIAL CELL INJURY:

Both Vasospasm and Endothelial cell injury are interlinked. *"Graudmann and associates"* reported that [circulating endothelial cells-CEC's] are elevated fourfold in the peripheral blood . *"Petrozella and colleaugues"* has reported increased levels of {circulating endothelial microparticles-EMP'S} in preeclamptic women.

Nitric oxide(NO): This is a potent vasodilator synthesized from L-arginine by endothelial cells. Reduced levels of NO "increases mean

arterial pressure, decreases heart rate and reverses the pregnancy induced refractoriness to vasopressors".

Endothelins: These are 21-amino acid peptides that are strong vasoconstrictors. Of these, plasma ET-1 level levels are increased more than the normal in preeclampsia.

Angiogenic factors:

Placental vasculogenesis is seen after 21 days of conception.Variety of angiogenic factors are implicated in the pathogenesis of preeclampsia. However, Angiogenic imbalance is the main cause. Apart from VEGF and Angiopeptins, Trophoblasts of women who are prone to preeclampsia overproduces mainly two anti-angiogenic peptides into the maternal circulation{described by *Karumanchi,2014}*namely,

1. " Soluble factor variant antigen",a variant of the Flt-receptor for PIGF[placental growth factor] & VEGF.It's level is increased that inactivate and decrease the circulating free PIGF & VEGF- endothelial dysfunction.Haggerty and colleagues found these levels are more markedly elevated in the II Trimester.

2. 'Soluble endoglin(sEng)'-also called as CD105 is a placenta-derived 65kDa molecule which blocks 'endoglin',{surface co-receptor for the TGF-beta family}. *Levin(2006) &Venkatesha(2006)*,quoted that this

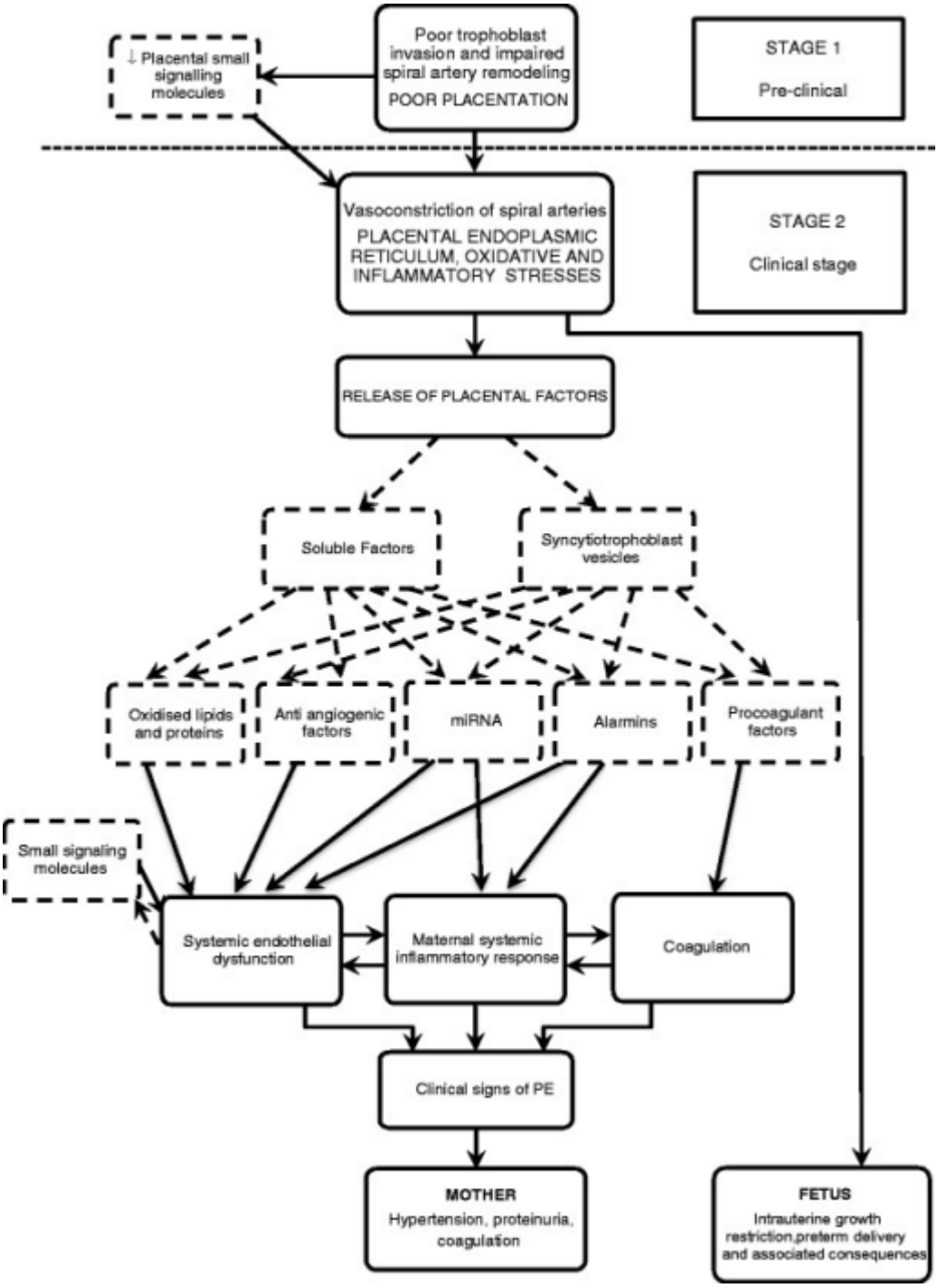
protein inhibits various TGF-beta isoforms from binding to the endothelial receptors causing decreased endothelial NO-dependent vasodilatation.It's level increases even before preeclampsia sets in.

PROSTAGLANDINS:

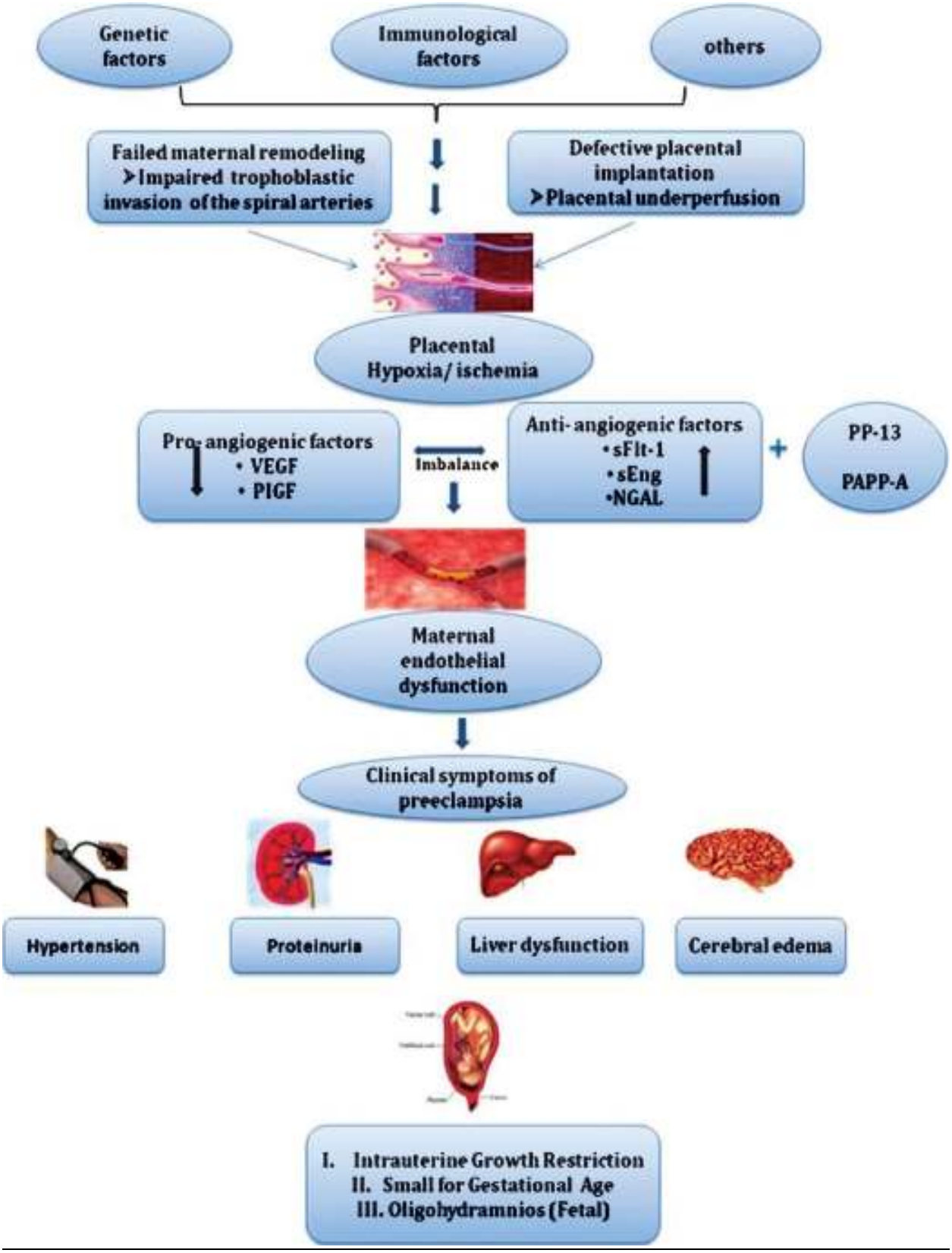
Endothelial prostacyclin(PGI2) is decreased in preeclampsia & Thromboxane A2 secretion is increased ⟹ increased sensitivity to ⟹ infused angiotensin II vasoconstriction.

The above angiogenic factors are directed to predict the severity of preeclampsia in the future years.

Finally, Haggerty and co-workers(2012) reported "doubling the expression of sFlt-1 & sEng increased preeclampsia by 39% and 74% respectively".



Schematic diagram showing the pathogenesis of PREECLAMPSIA.


Genetic factors
Immunological factors
others
Failed maternal remodeling
➢Impaired trophoblastic invasion of the spiral arteries
Defective placental implantation
➢Placental underperfusion
Placental Hypoxia/ ischemia
Pro- angiogenic factors
• VEGF
• PIGF
Imbalance
Anti- angiogenic factors
• sFlt-1
• sEng
•NGAL
+
PP-13
PAPP-A
Maternal endothelial dysfunction
Clinical symptoms of preeclampsia
Hypertension
Proteinuria
Liver dysfunction
Cerebral edema
I. Intrauterine Growth Restriction
II. Small for Gestational Age
III. Oligohydramnios (Fetal)

IMMUNOLOGICAL & GENETIC FACTORS:

Maternal immune tolerance to the paternally derived placental and fetal antigens is essential. “Loss of this TOLERANCE” results in pih as said by *Erlebacher(2013).* This dysregulation is due to

1.immunization from a previous pregnancy.

2.immunization from a some inherited human leukocyte antigen(HLA),NK cell receptor haplotypes,and shared susceptible genes.

CANDIDATE GENES *commonly involved are “MTHFR(C6771-Methylene tetrahydrate folate reductase)”F5(Leiden), AGT(M23(5T-Angiotensinogen), NOS3(Glu 298 Asp-endothelial nitric oxide), F2(G20210A), [CTLA4], ACE (I/D intron 16), LPL[lipoprotein lipase], SERPINE 1{ serine peptidase inhibitor}”*

PLACENTAL FACTORS:

Preeclampsia is associated with hyperplacentosis or abnormal placentation. Hence beta subunit of HCG{Human chorionic gonadotropin}which reaches its peak by 100 days after ovulation declines after that normally i.e., upto 12 weeks of gestation . But in preeclampsia, there is no decline in beta hcg values, in many studies recently as mentioned below(It raises or even mean/standard deviation of beta hcg

rises more than the normal when compared with the normotensive patients).

NUTRITIONAL FACTORS:

Zhang and associates(2002) mentioned that "the incidence of preeclampsia was doubled in women in whom the daily intake of ascorbic acid was

<85 mg/dl".Studies were conducted upon this. But according to "TASK FORCE STUDY 2013- in several trials, supplementation with the antioxidants vit.C or E showed no benefits".

Apart from the above aetiologies, ALTERED LIPID METABOLISM also plays an important role in the pathogenesis.

Studies have shown that the circulating lipids namely, triglycerides, total cholesterol, HDL, VLDL, LDL are increased during normal pregnancy. These are essential for

- Increased cellular proliferation of the maternal uterine enlargement.
- Blood volume expansion.
- Fetal implantation.

- Formation of blood vessels in the uteroplacental area and for

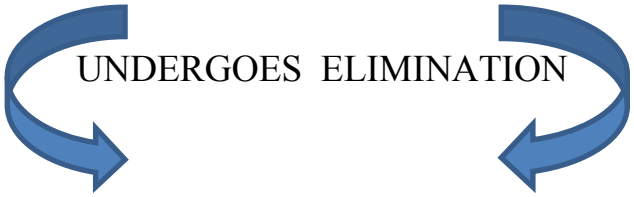
the fetoplacental development and growth.

HYPERHOMOCYSTINEMIA:

Homocysteine is an essential aminoacid required for the growth of the cells and tissues in the human body.

Homocysteine

Catalysed by Vit.B12 UNDERGOES ELIMINATION Catalysed by Vit.B6 and folic acid.



CYSATATHIONINE METHIONINE

Homocysteine is found in low concentrations in all tissues normally. It's level decreases with gestation due to

1.Physiological response to the pregnancy.

2.Decrease in Albumin.

3.Increase in Estrogen.

4.Hemodilution from increased plasma volume and

5.Increased demand for methionine by both the mother and the fetus.

Hyperhomcysteinemia is due to "mutations in methylene tetrahydrofolate reductase (MTHFR) gene, and cystathionine beta synthase(CBS) gene.

HYPERHOMOCYSTEINEMIA

↓

Increases the risk of atherosclerosis with oxidative damage.

↓

Homocysteine is oxidized to homocystine,homocystine mixed disulfids

↓

Formation of oxygen radicals and lipid peroxidation.

↓

Malondialdehydae- is a detector of lipid peroxidation.

Malondialdehyde is the end product of lipid peroxidation that leads to the oxidation of the biological system.

"*Steegens-Thenessi et al* presented that "hyperhomocysteinemia was associated with 2 to 3 fold increased risk of severe preeclampsia and eclampsia than mild preeclampsia".

" Increase in homocysteine levels → increase in oxidative free radicals due to lipid peroxidation → due to oxidation of biomolecules cellular dysfunction → causing maternal endothelial dysfunction → Leukocyte activation → end product formed –Malondialdehyde".

Also placental trophoblasts are rich in NADPH OXIDASE that are the source of free radical synthesis. Polyunsaturated free fatty acids are abundant in placenta causing lipid peroxidation → increased Triglycerides and LDL.

" *Ingec M et al* suggested that elevated levels of homocysteine are seen in severe preeclmpsia and eclampsia but not in mild preeclampsia".

In Preeclampsia, there is altered lipid metabolism leading to variation in lipid profile brought about by various studies as mentioned below.

STUDIES CONDUCTED:

I-STUDY:

A prospective study was conducted between 2008 and 2009 in the dept. of Obstetrics and Gynaecology, SMS medical college ,Jaipur, in the normotensive, non-proteinuric women between 13 to 20 weeks of gestation. This study included the antenatal patients irrespective of the parity.

In this study,200 women were involved and they were followed up till term gestation. Here out of 200 only 90 were in pih group where b-hcg was >2MoM,other 110 were normotensives .It concluded that there was no significant statistical association between the maternal age, parity, religion and preeclampsia, but increase in pih was more in primiparas.

There was increased association of beta hcg with preeclampsia-sensitivity being 90.91% and specificity of 97.44% & positive predicitive value-83.33%.

Desai & Rao studied that out of 90 cases,62{68.9%}showed b-hcg>2 MoM & 20 cases of 130 showed b-hcg <2MoM.

"Roiz –Hernandez et al" showed that sensitivity, specificity and positive predicitive value were 88.5%,100% &0.46 respectively when b-hcg >2MoM.)

Kabukcu et al., studied that 610 pregnant women in second trimester,grouping them according to Multiple of Median(MoM).He concluded that b-hcg>2MoM are at high risk for preeclampsia.

II- STUDY:

A prospective study was conducted in Midanapur medical college and Hospital ,West Bengal & Silchar Medical College, Assam between September 2010 to August 2011.Here, a total of 100 antenatal patients (opd patients) selected, of which 18 developed preeclampsia and 82 were normotensives and all were followed up till term.

b-hcg >=40,000 IU/ml was the cut off where the mean+- SD of serum b-hcg was significant(p-value<0.0004),whereas the mean+- SD of the blood pressures between the pih and the normotensive group does not vary except only for high diastolic pressures.

The LIPID PROFILES namely TC,,Triglycerides values were significantly increased in pih patients. [i.e. for 1 unit increase in total

cholesterol and triglycerides,there is a 0.3% of the pregnant women developing PIH- P-value-0.0154].Whereas HDL and LDL values rise shows a mild decrease in pih i.e.{for 1 unit increase in HDL, there is a 7% less chance of developing pih & for 1 unit of LDL rise, there is a 6.6% less chance of developing pih}. But for 1 unit of VLDL rise ,there is 29.8% developing pih.

With respect to b-hcg, weight and age,there is a significant relationship in pih,{ each 1000mIU/ml rise in b-hcg, there is 20.8% chance of developing pih}.

By binomial logistic regression analysis, 1 kg increase in weight, decreases the risk of pih by 30.3%.

As the age advances by one year, there is 14.4% less chance of incidence in pih & as the parity increases, there is 123% greater risk of developing pih.

Hence , this study concluded that there was significant relationship between b-hcg, weight, age, parity, TG, VLDL, triglycerides, blood pressure, LDL, HDL and preeclampsia [b-hcg, triglycerides, VLDL, age, weight, blood pressure rise affect pih more than HDL ,VLDL]

III-STUDY:

A prospective case study was done at Shree Sayaji General Hospital, Baroda from 1st April 2011 to 31st March 2012.This study was conducted in 110 antenatal patients,out of which only 100 patients were on regular follow up.

Among the 100 cases only 26 developed pih, in which 18 developed severe preeclampsia, 6 developed gestational hypertension, and 2 developed eclampsia.

The cut off of b-hcg in this study developing pih was 40,000mIU/ml and above. Only 20 out of 26 (77%) patients had this value(P< 0.000) with the prevalence rate of 87%, whereas, *Vidyabati R K et al* showed the above statistics with the prevalence rate of 95%.

On the lipid profile abnormalties,18/26 had total cholesterol >200mg/dl(34.6 %), 9/26 had triglycerides >=150mg/dl{P<0.000 &P<0.005) respectively. Among the 100 ,irrespective of those developing pih, 33/100 had total cholesterol > 200 mg/dl,13 had HDL<40 mg/dl,15 had LDL>130 mg/dl,12 had VLDL>35 mg/dl.

In respect to the age and parity, out of 26 patients, 11 were between the age group of 18 to 21 years,rest 15 belong to more than 21 yrs & pih

was more in primiparas. In this study, body weight and BMI were not considered significant.

Finally, this study concluded that out of 100 patients studied, 47% were primi, 20 out of 26 had b-hcg > 50,000mIU/l ,18 out of 33 had high cholesterol levels, suggested dyslipidemia and elevated serum beta hcg levels are very good noninvasive predictors of early second trimester pih.

IV-STUDY:

Another prospective study conducted by Maharaja Agrasen Hospital, Punjabi Bagh, New Delhi, from March 2010 to March 2011.This study included 120 antenatal patients of the outpatient department and followed up till delivery. All were screened for serum beta hcg and serum lipid profile taking the blood sample after 12 hrs fasting.

In this study out of 120, only 21 cases developed pih and the remaining 99 were normotensives and the prevalence was 17.5%.However,*Vidyabati et al* found prevalence as 17.68% in about 164 cases in this study.

The statistical data analysis are as follows;

42.9% - triglycerides> 200 mg/dl.

71.4% - total cholesterol>200mg/dl;

47.6% - HDL>40 mg/dl;

47.6% - LDL>130mg/dl;all the above had p-values <0.0005.

Whereas, 19/21cases developing pih showed HDL

90.5% - HDL >65 mg/dl. And of 99 normotensive cases ,

89/99 had the similar hdl levels- >65mg/dl in 89.90% of patients.

There was no significant rise in beta hcg observed in this study comparable between the pih and normotensive group.

With respect to age, parity, BMI, Blood pressures,

51.7% - age group between 25-29 yrs,only 3 were > 35 years,

Based on parity, 59 cases were primi (57.84%), 61 were multigravida (57.14%),

Out of 21 cases in the pih group,12 were primi.(majority).Almost all the cases were of BMI between 20-25 kg/m2 except six cases (>30 kg/m2- 2 developed PIH and 4 were normotensive).

With respect to Blood pressure, both systolic and diastolic blood pressure were higher in pih {group-II} when compared to normotensive{group –I}.

Mossink et al and Pouta et al concluded that "rise in b-hcg was not observed in gestational hypertension cases but only in severe preeclampsia",

Lorentzen et al concluded that "serum- free fatty acids and triglycerides were increased before 20 weeks of gestation and later they developed preeclampsia."

Cekemen et al showed that " plasma triglycerides &LDL were higher in preeclampsia,whereas HDL were lower in preeclamptic patients".

De et al concluded that " triglycerides and VLDL were raised and HDL levels decreases in preeclamptic patients".

Vidyabati et al showed that "total cholesterol,VLDL,LDL were significantly raised in preeclampsia patients".

Hence this study concluded that there is no significant relationship between HDL, serum b-hcg and pregnancy induced hypertension whereas other parameters are elevated in preeclamptic group-II than normotensives (group-I).

V-STUDY:

This study was conducted in the Regional Institute of Medical Sciences, Imphal between Novemeber 2005-December 2006.

A total of 164 out of 180 (16 Patients were not on regular follow up)patients {including the antenatal opd patients & ward patients between 14 to 20 weeks of gestation not involving the exclusion criteria}were included in this study.

Of these ,29 cases developed pih and the remaining 135 were normotensives.

Mean +- SD of all the parameters were calculated for pih and normotensive patients and the difference of means were tested by t-test.A higher statistical formula- Multiple Logistic Regression Model was used to estimate the causal effect of each predisposing factor on response variables. Their effect are measured in terms of Odds Ratio.

The serum b-hcg was significantly increased($P<0.000$)in those women who developed pih. Total cholesterol and VLDL were significantly increased($P<0.0000$ & $P<0.027$)who subsequently developed pih.

Mulitple Logistic Regression analysis showed that of every 1000 mIU/L increase in serum b-hcg, a pregnant women has 10.7% increased

chance of developing pih.For 1 unit increase in TC,TGL,VLDL.LDL, there are 12.6%,0.3%,12.4%,7.1% of increased incidence of developing pih.

On the contrary, 1 unit of increase in HDL, there is 11.4% less chance of developing pih.

Of 29 cases,21 had elevated serum b-hcg & 28 had dyslipidemic parameters

However, there is significant rise in blood pressure(both systole and diastole) at the time of delivery in pih patients than normotensive. Through MLR variable analysis, one kg increase in weight, controlling other variables ,there is 12.7% chance of developing pih, each year advancing age increases pih by 9.95%, for one unit increase in Hb% ,there is 28.8% decreased chance of developing pih.

With respect to parity, according to this study, 52% occurs in elderly primi(31-35 years) and the mean age+- SD was[27.17+-4.08].

Mean +-SD of the HDL values in both the groups were found be similar with no significant association to pih.

This study concluded that serum b-hcg and TG, TC, LDL, VLDL, weight, age are associated with the increased incidence of pih if these are elvated more than the mean+-SD.

And HDL & Hemoglobin rise leads to a fall in preeclampsia in the early second trimester.

VI-STUDY:

Another prospective study was done at Department of Pathology and Obstetrics and Gynaecology, VMMC, Safdarjung Hospital, MGH hospital at New Delhi between January 2011 and December 2012.

This study included only a small community of the antenatal opd patients. Only 55 patients were selected, out of which 25 developed pih and the remaining 30 were normotensives.

With respect to age, parity ,about 48% of patients fall between 25-29 years in pih group and 33% fall in the normotensive group.By parity wise,

16/25 patients - primigravidas accounts to 54.54% of the population.

14/30 patients - primigravidas (Normotensives) accounts to 45.45% of the Population.

Blood pressure{both systole and diastole} rise is observed in this study in pih patients than healthy controls($p<0.05$).

The levels of beta hcg in the study group{41,500} that was significantly higher than the control group($p<0.0001$).

The serum cholesterol levels(218.4),LDL(136mg/dl) & URIC ACID levels (6.3) are also elevated in pih.

However,there was no statistical association between HDL, VLDL, triglycerides and preeclampsia.

OBSERVATIONS & RESULTS.

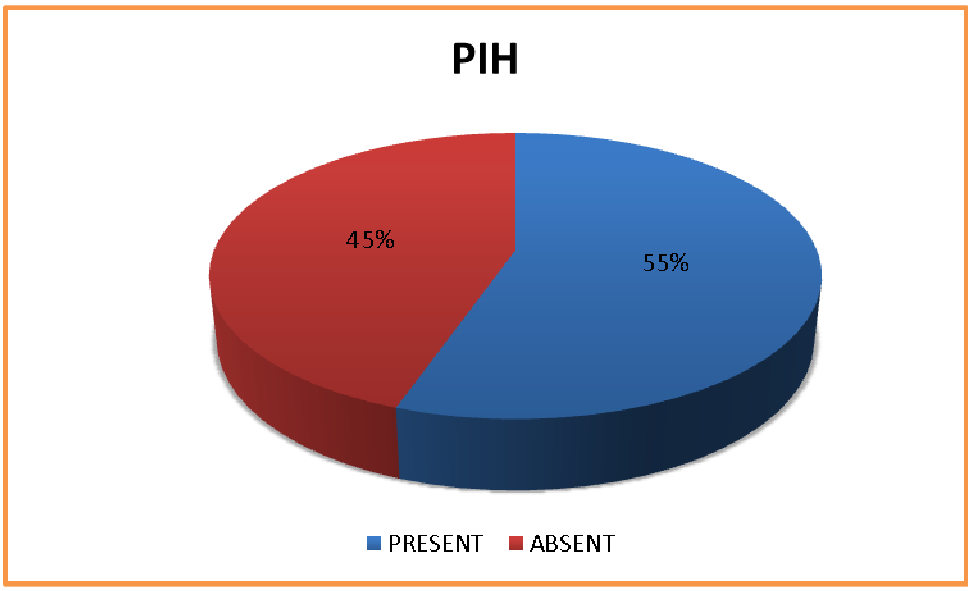
AGE- It's relation to pih.

PIH	NO OF PATIENTS	PERCENTAGE
PRESENT	83	55%
ABSENT	67	45%

The above table depicts that in our study, out of 150 patients,

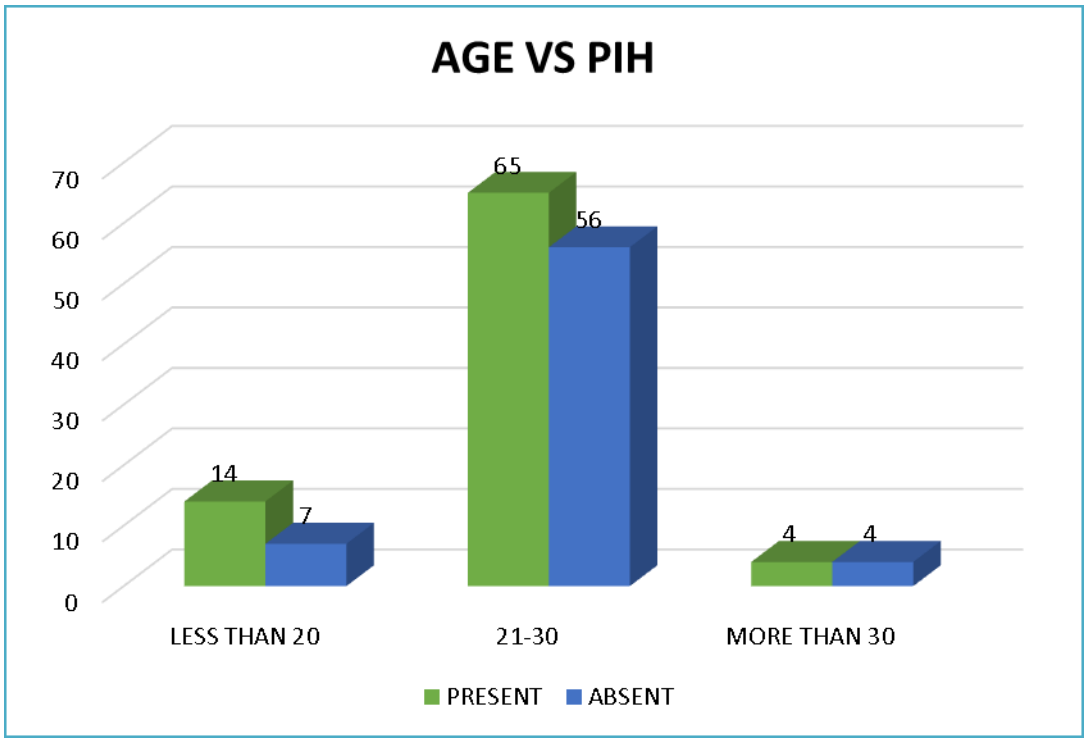
83cases - study group(preeclamptic) accounts for 55% of the sample.

67cases -normotensives represents 45% of the sample.



The Table describes that of the 150 cases, 121 falls in the age group between 21 to 30 yrs , remaining 29 fall in either category(Bar chart depicts it.)

AGE (IN YEARS)	NO OF PATIENTS	PERCENTAGE
LESS THAN 20	21	14%
21-30	121	81.00%
MORE THAN 30	8	5%

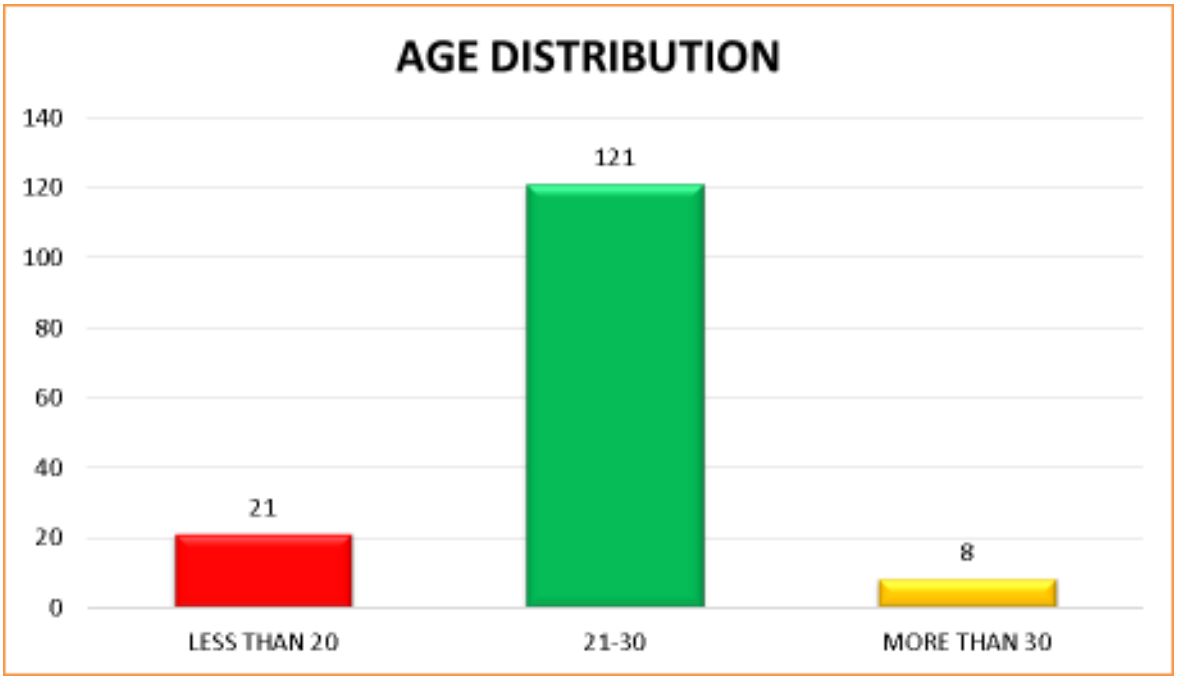


The below table represents that between the age group of 21 to 30 yrs , and in extremes <20 yrs & >30 yrs, half of them are normotensive & other half are pih.

	PIH	
AGE (IN YEARS)	PRESENT	ABSENT
LESS THAN 20	14	7
21-30	65	56
MORE THAN 30	4	4
P VALUE - 0.519		

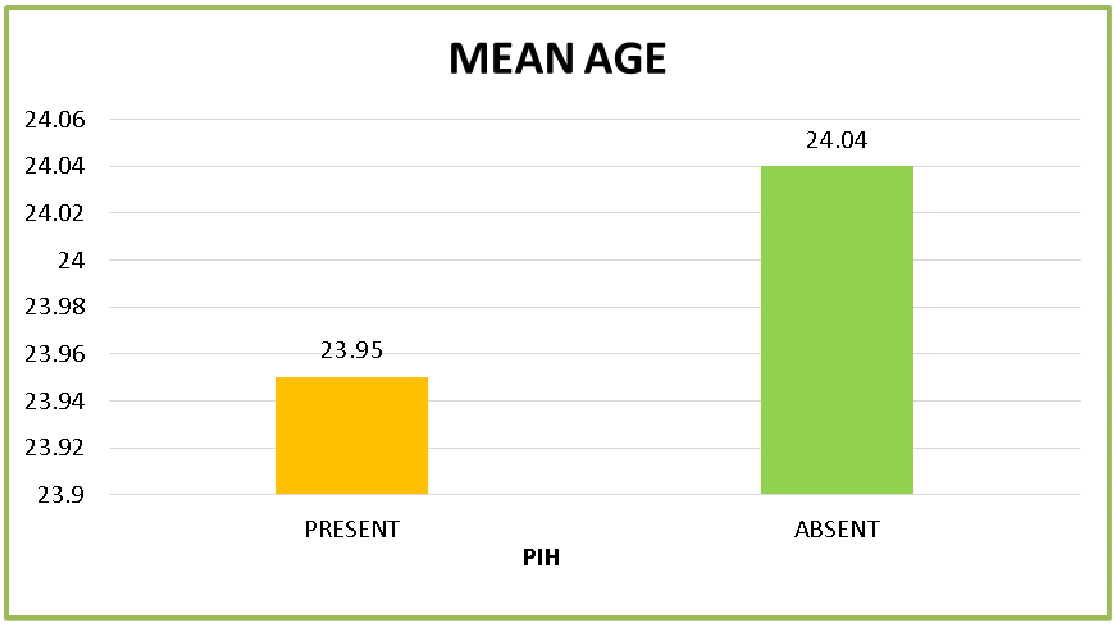
NON SIGNIFICANT

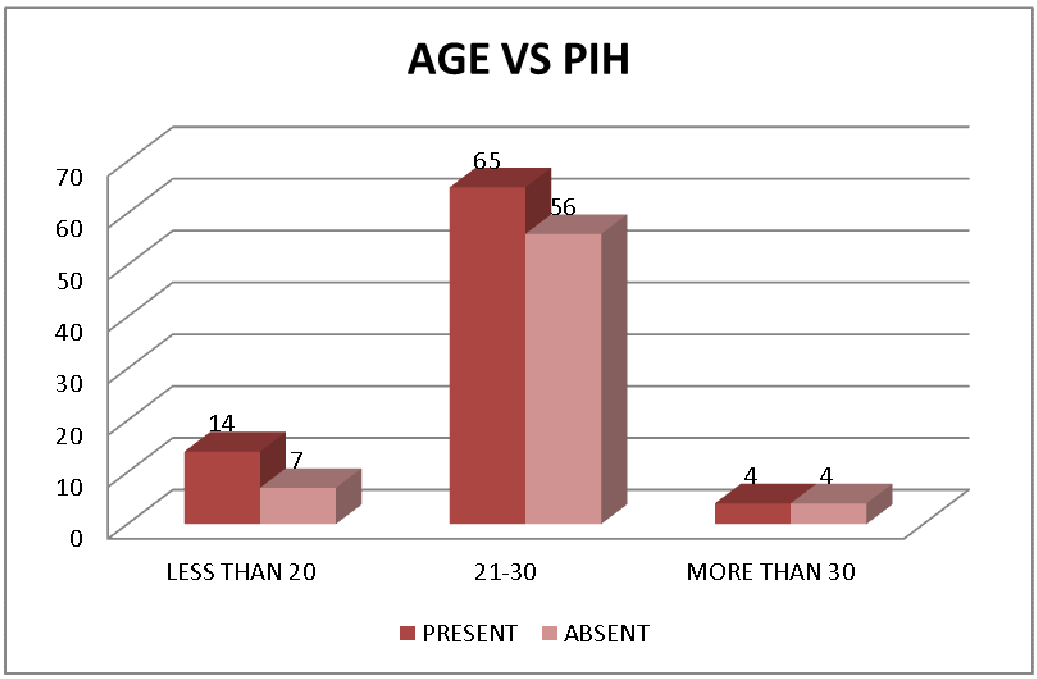
KRUSKAL WALLIS TEST



	AGE	
PIH	MEAN	S.D
PRESENT	23.95	3.68
ABSENT	24.04	3.9
UNPAIRED T TEST		
P VALUE - 0.832		

The mean +- SD of the age is not significantly related to pih(p-value-0.832) as depicted below.





The above bar diag. represents the age presentation in the 150 patients.

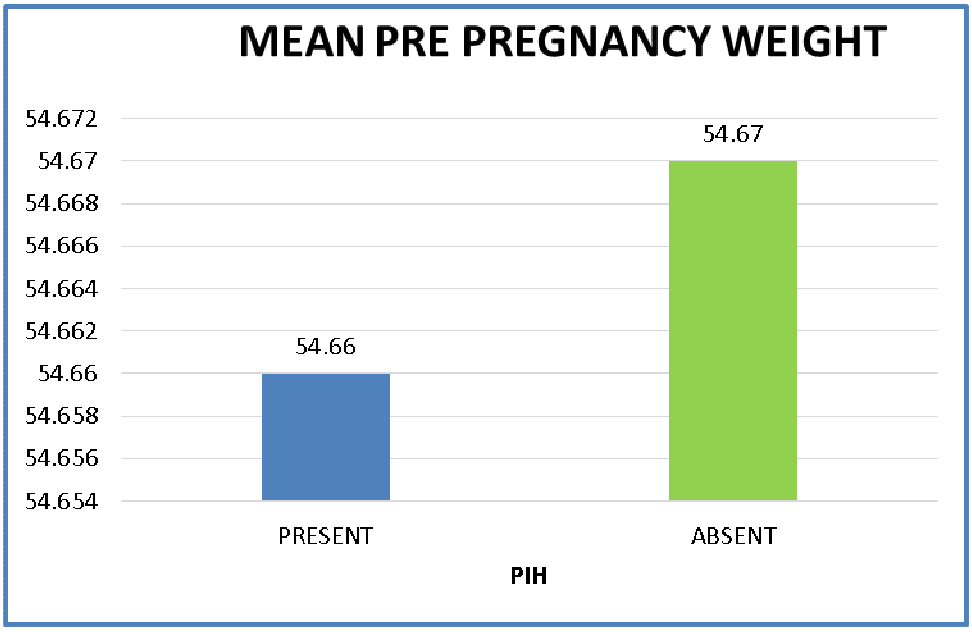
WEIGHT-It' relation to pih

The weight increases significantly from the pre-pregnancy range to the present weight where P-value <0.0001. But there is no significant relationship between the present weight and incidence of preeclampsia.

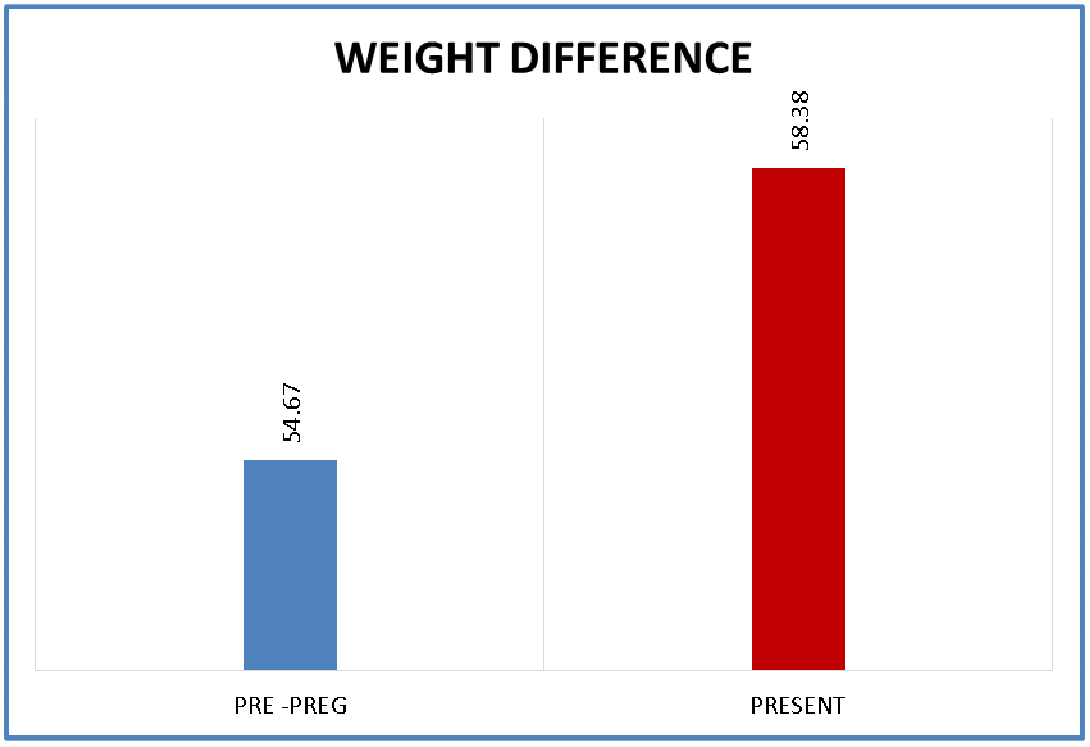
WEIGHT	MEAN	S.D
PRE -PREG	54.67	8.5
PRESENT	58.38	8.34
P VALUE - 0.001		
SIGNIFICANT		
PAIRED T TEST		

Pre-pregnancy weight:

	PRE PREGNANT WT	
PIH	MEAN	S.D
PRESENT	54.66	8.29
ABSENT	54.67	8.8
UNPAIRED T TEST		
P VALUE - 0.995		
NON SIGNIFICANT		

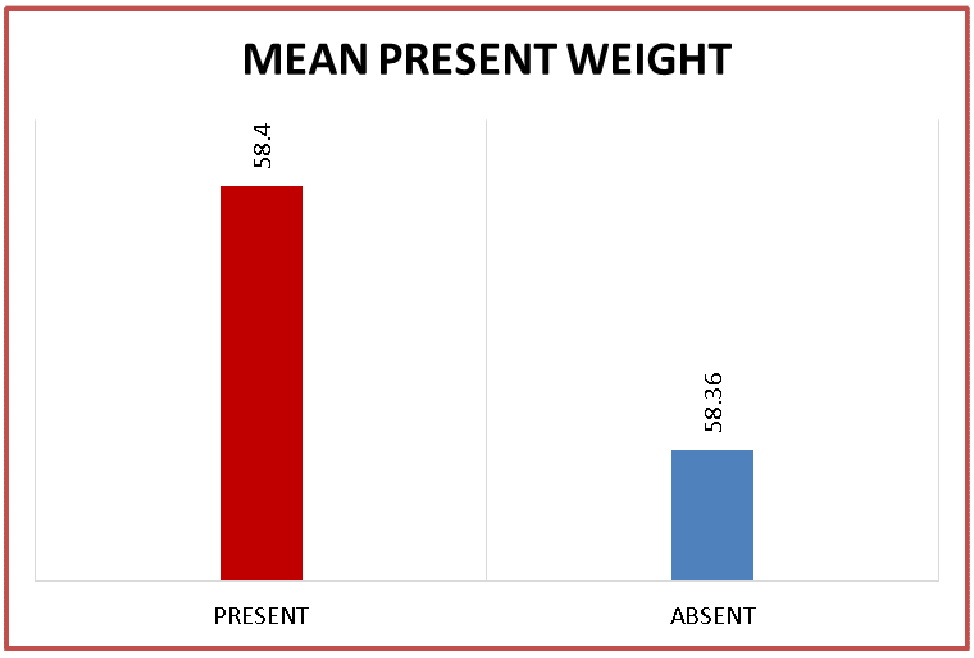


The above bar diagram depicts that there is no significant relation between pre-pregnant weight and pih.(p-0.995).



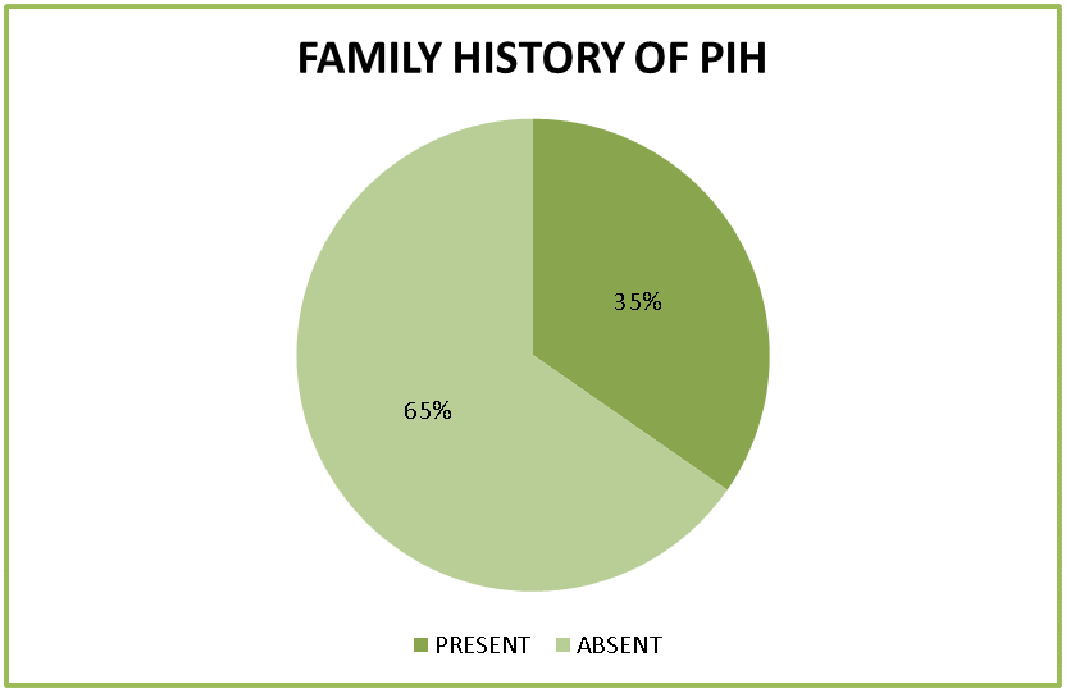
	PRESENT WEIGHT	
PIH	MEAN	S.D
PRESENT	58.4	8.1
ABSENT	58.36	8.6
UNPAIRED T TEST		
P VALUE - 0.977		
NON SIGNIFICANT		

The above table depicts that the mean +- SD of the present pregnancy weight is not significantly related to the preeclampsia.(p-value-0.977).



FAMILY HISTORY- It's incidence in pih

	NO OF PATIENTS	PERCENTAGE
PRESENT	52	35%
ABSENT	98	65%



By chi-square test, there is no relation between family history and preeclampsia.

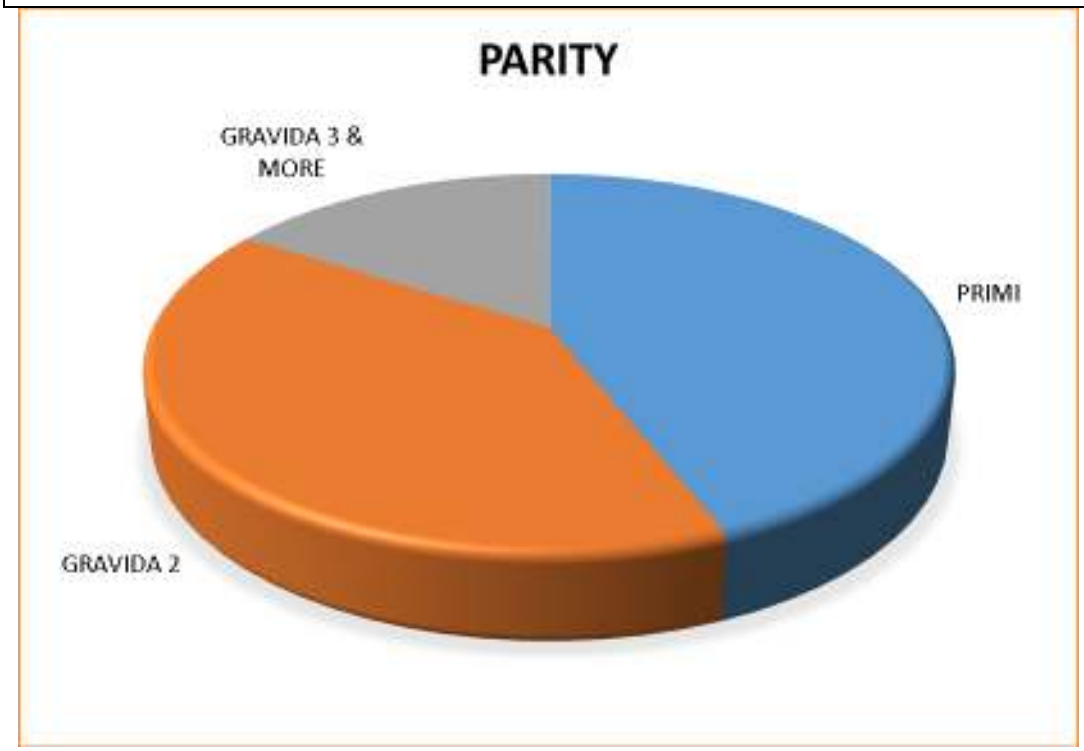
	PIH	
FAMILY H/O	PRESENT	ABSENT
PRESENT	38	19
ABSENT	50	48
P VALUE - 0.145		

PARITY:

PARITY	NO OF PATIENTS	PERCENTAGE
PRIMI	66	44%
GRAVIDA 2	60	40%
GRAVIDA 3 & MORE	24	16%

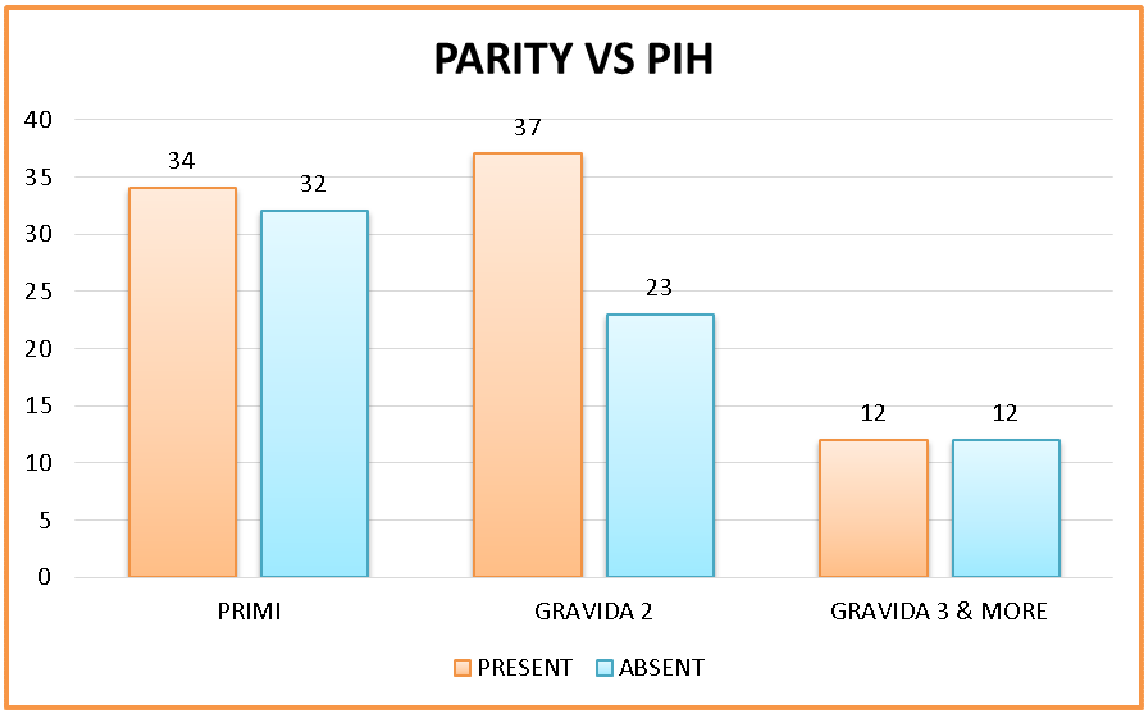
	PIH	
PARITY	PRESENT	ABSENT
PRIMI	34	32
GRAVIDA 2	37	23
GRAVIDA 3 & MORE	12	12
P VALUE - 0.441		

NON SIGNIFICANT
CHI SQUARE TEST



The above picture depicts that out of 150 samples , majority of them are (primigravida included in the study >G2>G3)

In my study,{incidence of preeclampsia is not more in primi when compared to other studies}.

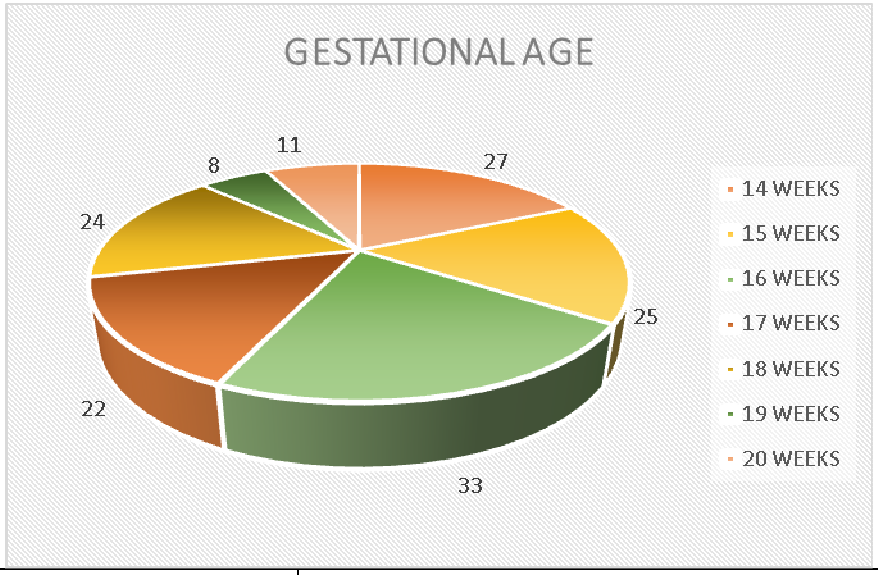


GESTATIONAL AGE.

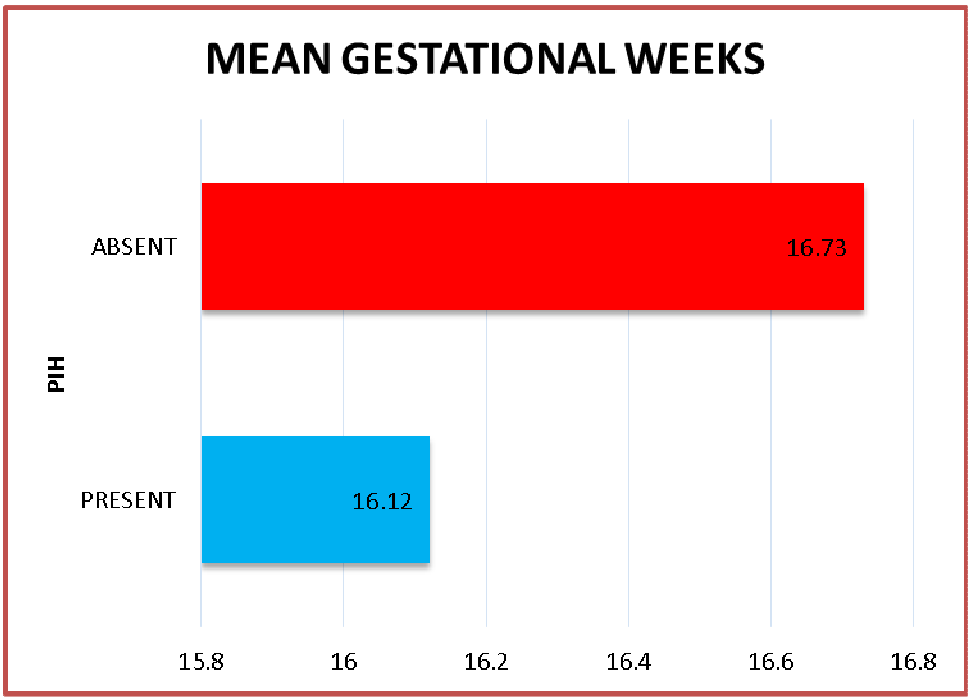
In this study, the patients with 16 weeks of gestation form 22% of population with

GESTATIONAL AGE	NO OF PATIENTS	PERCENTAGE
14 WEEKS	27	18%
15 WEEKS	25	16.70%
16 WEEKS	33	22%
17 WEEKS	22	14.70%
18 WEEKS	24	16%
19 WEEKS	8	5.30%
20 WEEKS	11	7.30%

The least being the 19 weeks of gestation.



	GESTATIONAL WEEKS	
PIH	MEAN	S.D
PRESENT	16.12	1.7
ABSENT	16.73	1.8
UNPAIRED T TEST		
P VALUE - 0.037		
SIGNIFICANT		



The mean and SD in relation to the gestational age signifies that at 16 weeks of gestation the incidence of pih is increased.(p-value-0.037).

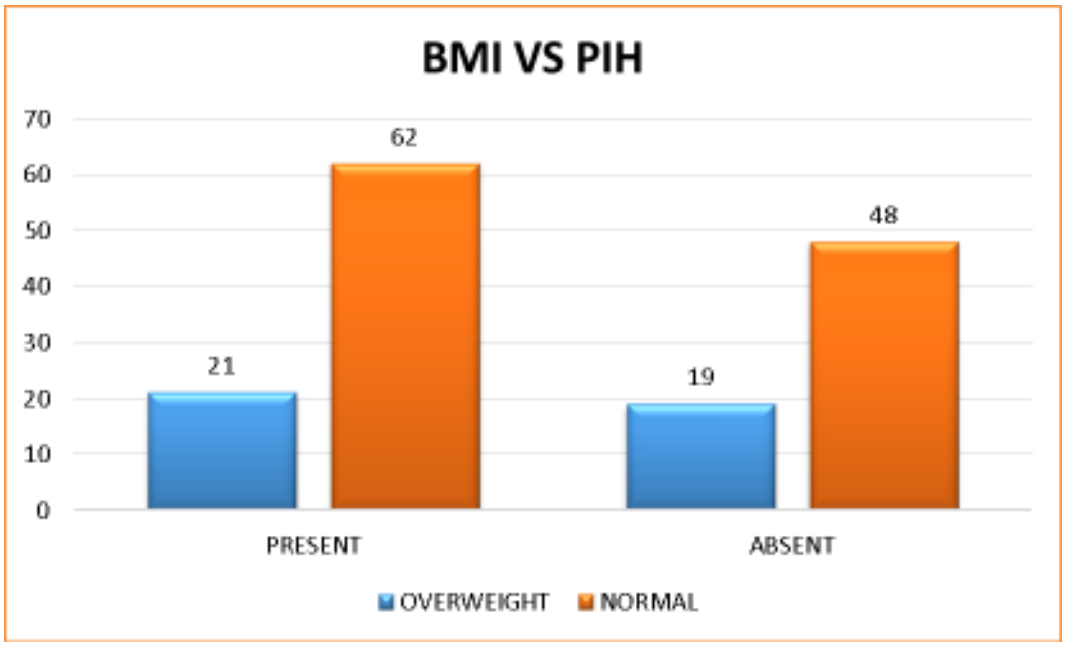
BODY MASS INDEX	NO OF PATIENTS	PERCENTAGE
NORMAL	110	73%
OVERWEIGHT	40	27%

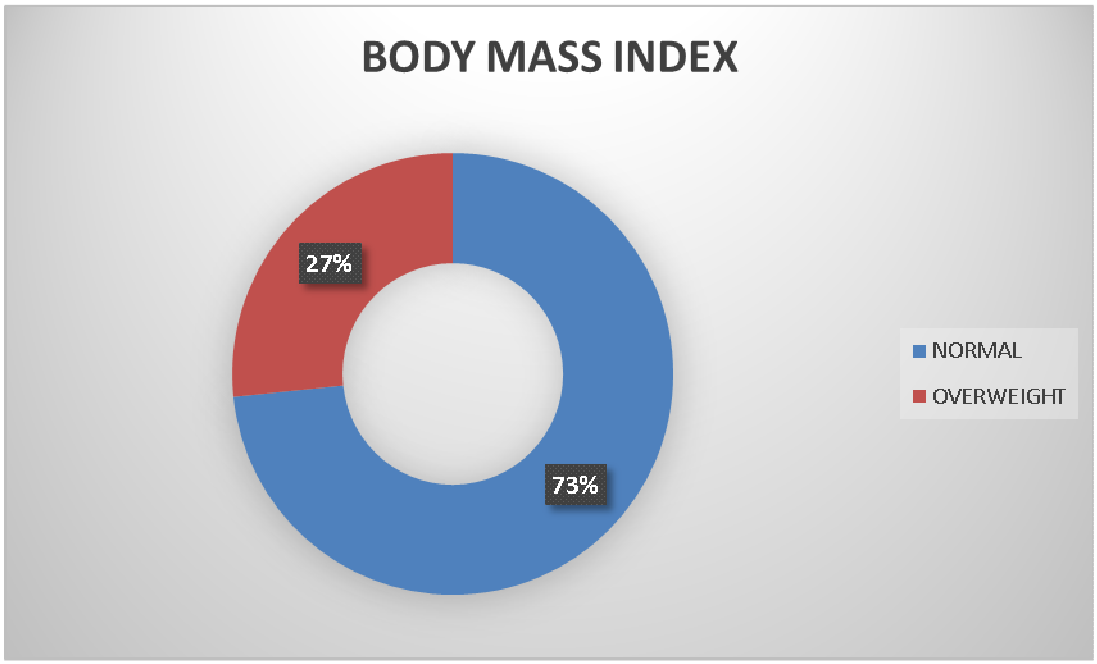
BODY MASS INDEX and its relation to PIH.

In my study, out of 150 patients, 110 were with normal BMI. The remaining 40 were overweight (bmi>30kg/m2).

	PIH	
BODY MASS INDEX	**PRESENT**	**ABSENT**
OVERWEIGHT	21	19
NORMAL	62	48
P VALUE - 0.674		
NON SIGNIFICANT		
CHI SQUARE TEST		

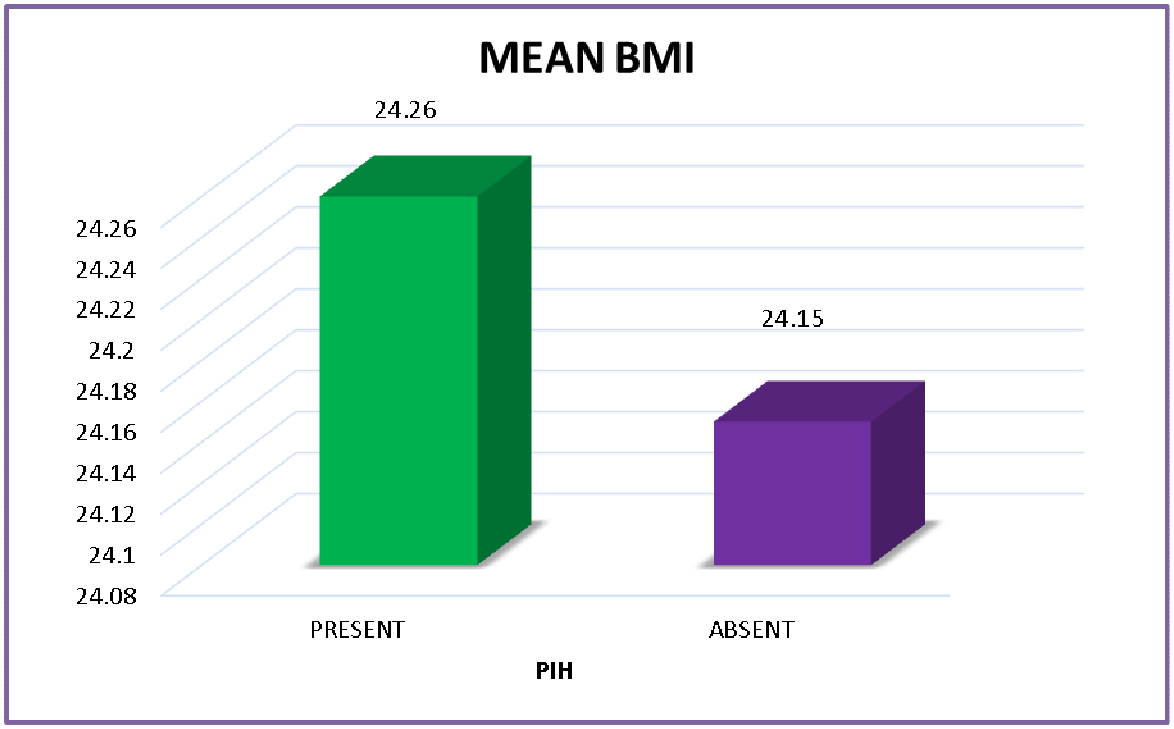
With respect to preeclampsia, out of 150 patients, 62 (normal BMI) & 21(BMI>30)i.e.{there is no significant relation between BMI and PIH)in my study.





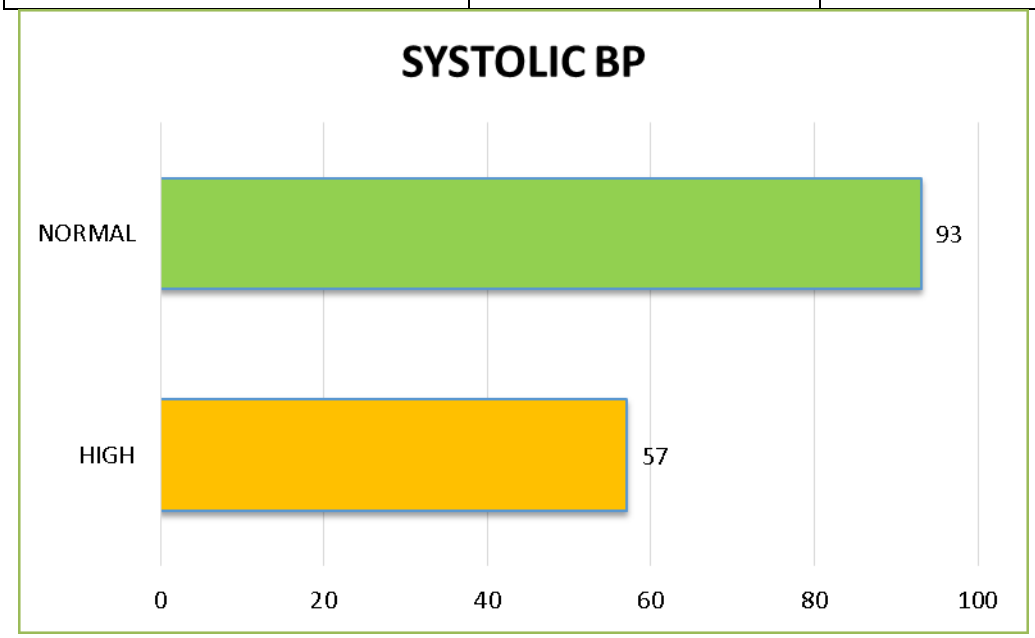
This diag. depicts that 73/150 patients have normal BMI.

	BODY MASS INDEX	
PIH	MEAN	S.D
PRESENT	24.26	1.27
ABSENT	24.15	1.02
UNPAIRED T TEST		
P VALUE - 0.105		
NON SIGNIFICANT		



BLOOD PRESSURE SIGNIFICANCE:

SYSTOLIC BP	NO OF PATIENTS	PERCENTAGE
HIGH	57	38%
NORMAL	93	62%



Out of 150 patients,only 57 had systolic blood pressure >140 mmhg (includes pih,mild and severe preeclampsia).

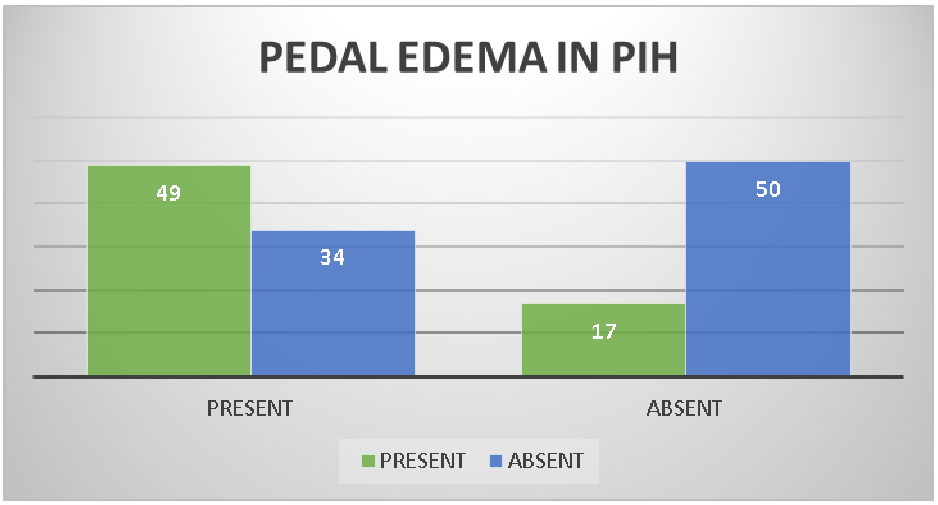
DIASTOLIC BP	NO OF PATIENTS	PERCENTAGE
HIGH	67	45%
NORMAL	83	55%

In this study, diastolic pressure is more in 67% patients of preeclampsia.

PEDAL EDEMA	NO OF PATIENTS	PERCENTAGE
PRESENT	66	44%
ABSENT	84	56%

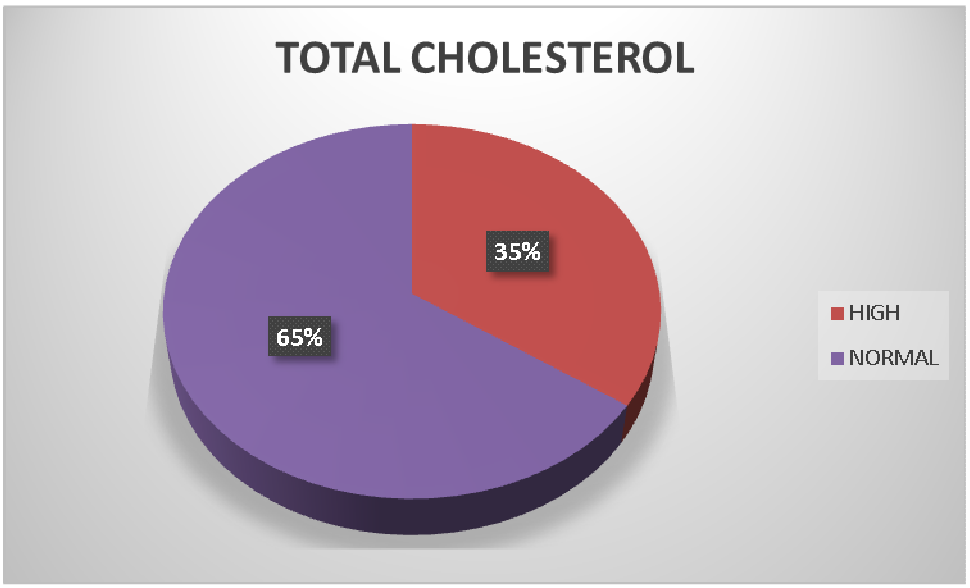
	PIH	
PEDAL EDEMA	PRESENT	ABSENT
PRESENT	49	17
ABSENT	34	50
P VALUE - 0.002		
SIGNIFICANT		
CHI SQUARE TEST		
ODD'S RATIO- 4.2		

Out of 150 ,66 had pedal edema & 84 does not have.However,49/66 presented with pih.remaining 17 were normotensive.Hence, there was a higher incidence of pih in patients with pedal edema.


PEDAL EDEMA IN PIH
49
34
17
50
PRESENT
ABSENT
PRESENT
ABSENT

TOTAL CHOLESTEROL	NO OF PATIENTS	PERCENTAGE
HIGH	52	35%
NORMAL	98	65%

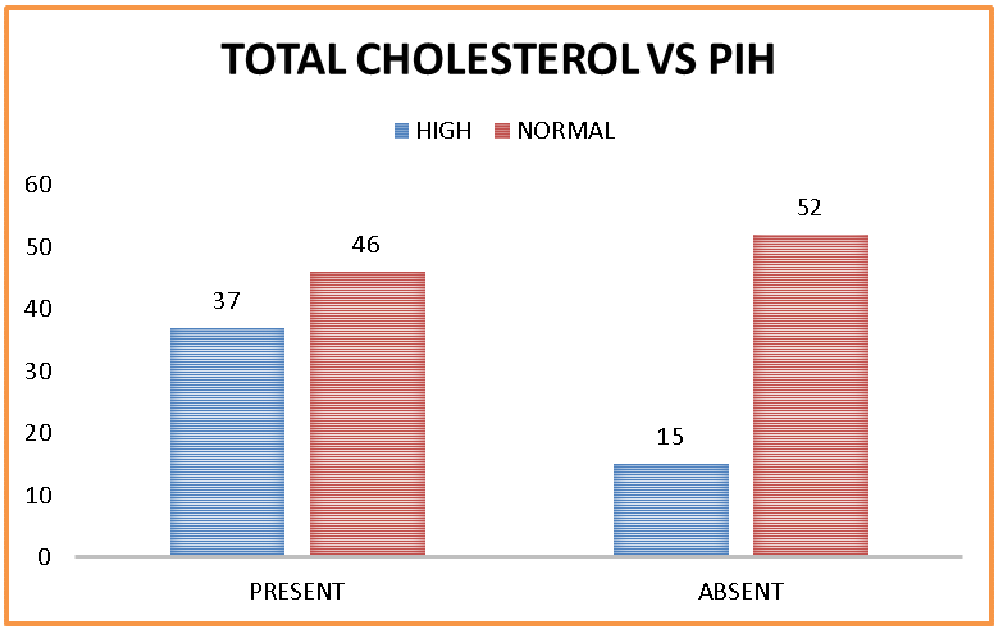
TOTAL CHOLESTEROL and its relation to PIH.



	PIH	
TOTAL CHOLESTEROL	**PRESENT**	**ABSENT**
HIGH	37	15
NORMAL	46	52
P VALUE - 0.005		
SIGNIFICANT		
CHI SQUARE TEST		
ODDS RATIO -2.78		

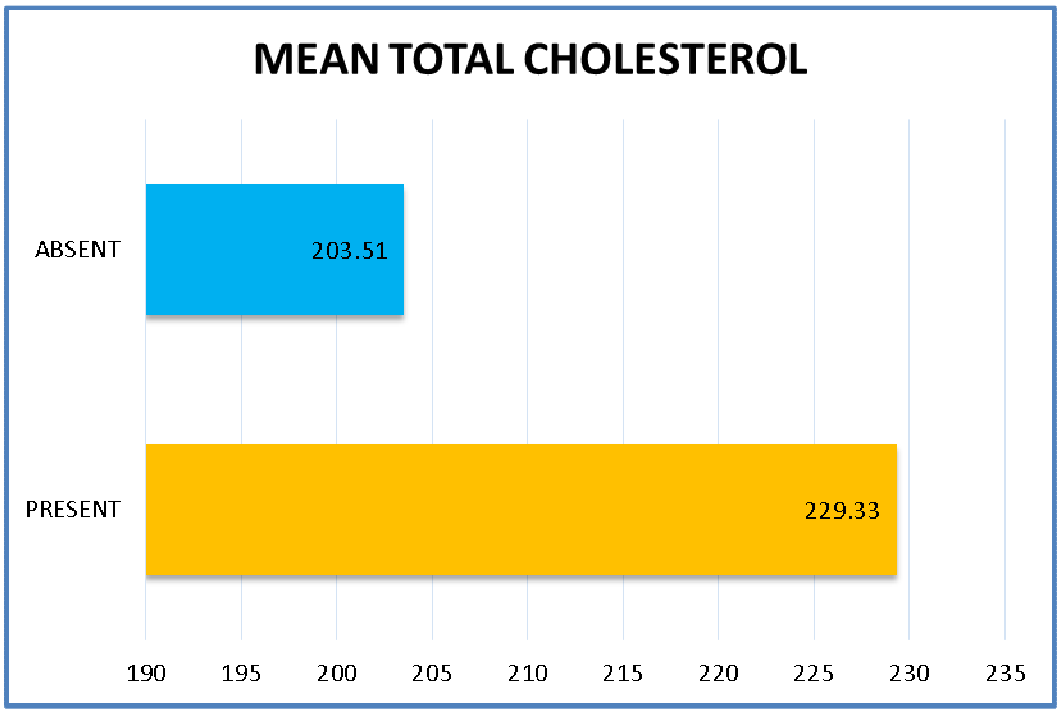
The total cholesterol level is increased in 46 patients out of 83 pih patients.

Whereas majority of the normotensives have normal cholesterol levels.



	TOTAL CHOLESTEROL	
PIH	**MEAN**	**S.D**
PRESENT	229.33	61.41
ABSENT	203.51	63.48
UNPAIRED T TEST		
P VALUE - 0.013		
SIGNIFICANT		

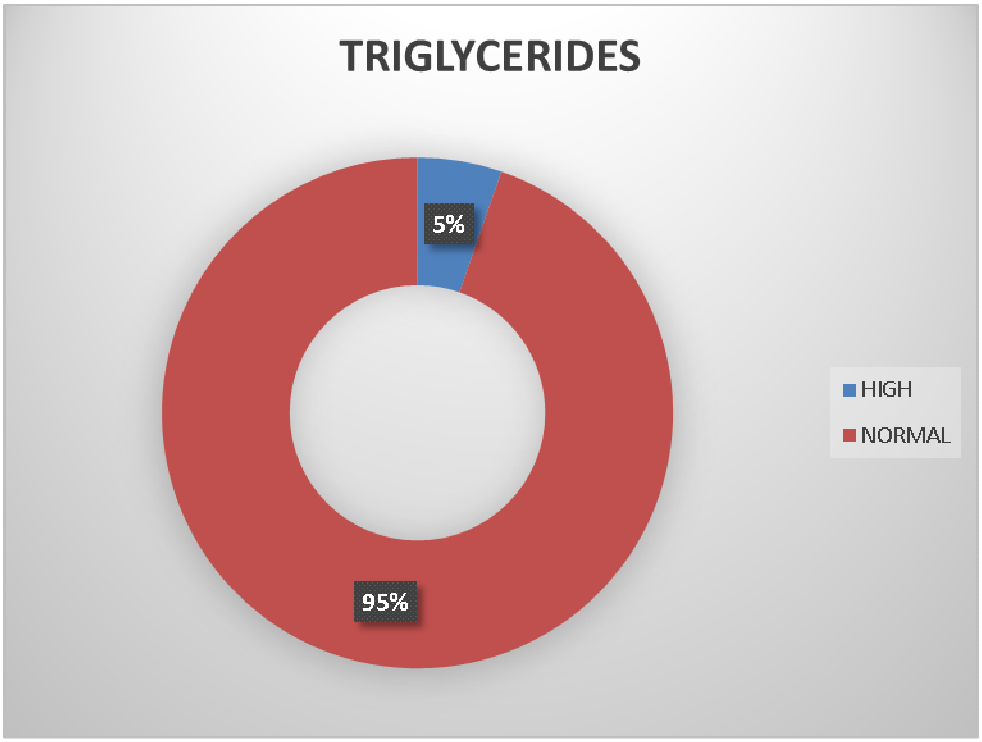
The Mean & S.D. of the total cholesterol is more than the normal and (p-value <0.013 that is significantly related to pih)

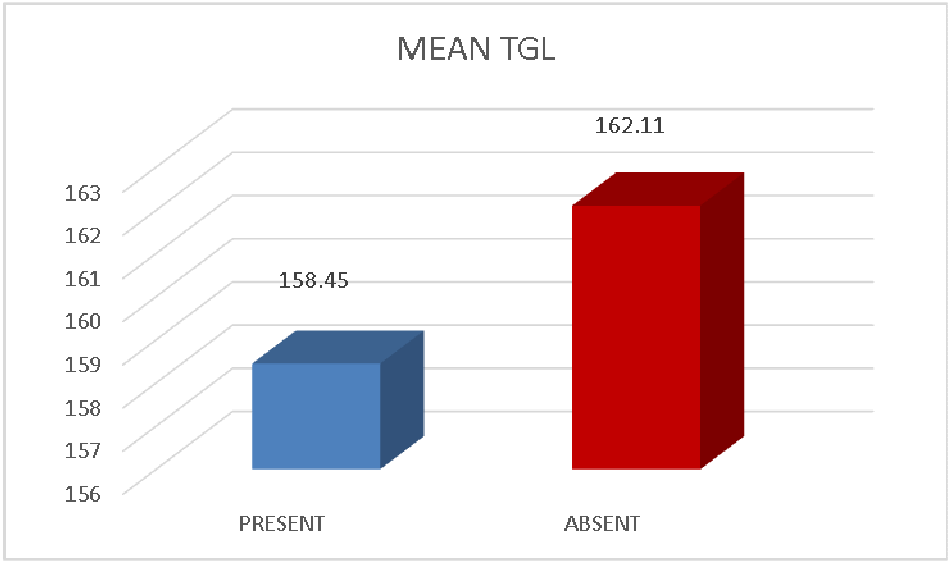


TRIGLYCERIDES	NO OF PATIENTS	PERCENTAGE
HIGH	8	5%
NORMAL	142	95%

TRIGLYCERIDES –It's relation to PIH.

The below table and diagram depicts that 95% of patients in the study had normal triglyceride levels.





	TRIGLYCERIDES	
PIH	MEAN	S.D
PRESENT	158.45	30.56
ABSENT	162.11	50.77
UNPAIRED T TEST		
P VALUE - 0.585		
NON-SIGNIFICANT		

The table illustrates that mean and S.D. of the triglyceride levels are below the nomal values in this study in pih patients (p-value –0.585) that is non-significant by unpaired t- test method.

HIGH DENSITY LIPOPROTEINS – It's relation to PIH.

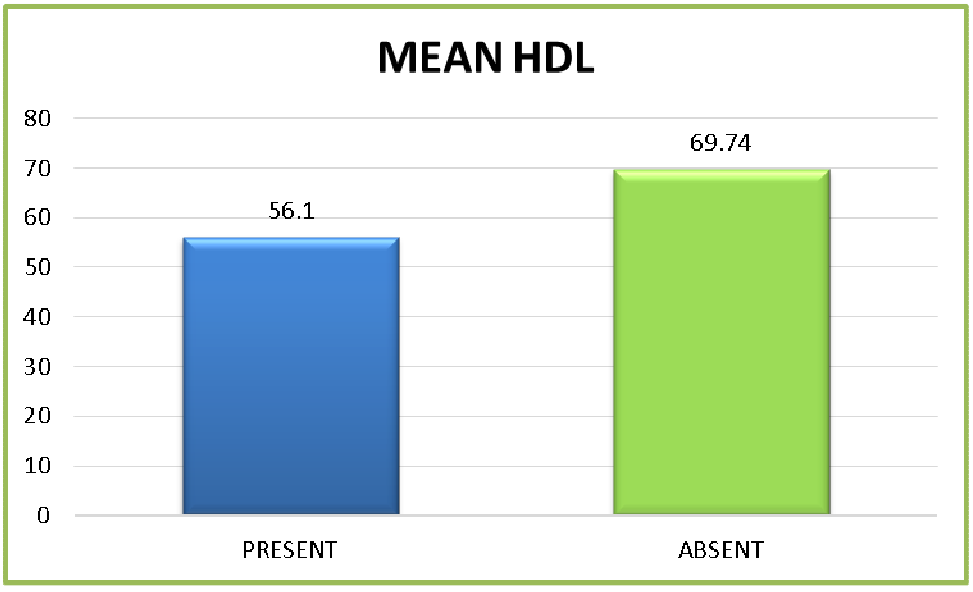
HDL	NO. OF PATIENTS	PERCENTAGE
LOW	58	39%
NORMAL	92	61%

HDL levels are normal or decreased in ¾ th of pih patients.(p-value-0.316).

	PIH	
HDL	**PRESENT**	**ABSENT**
LOW	44	30
NORMAL	39	37
P VALUE - 0.316		
NON SIGNIFICANT		
CHI SQUARE TEST		

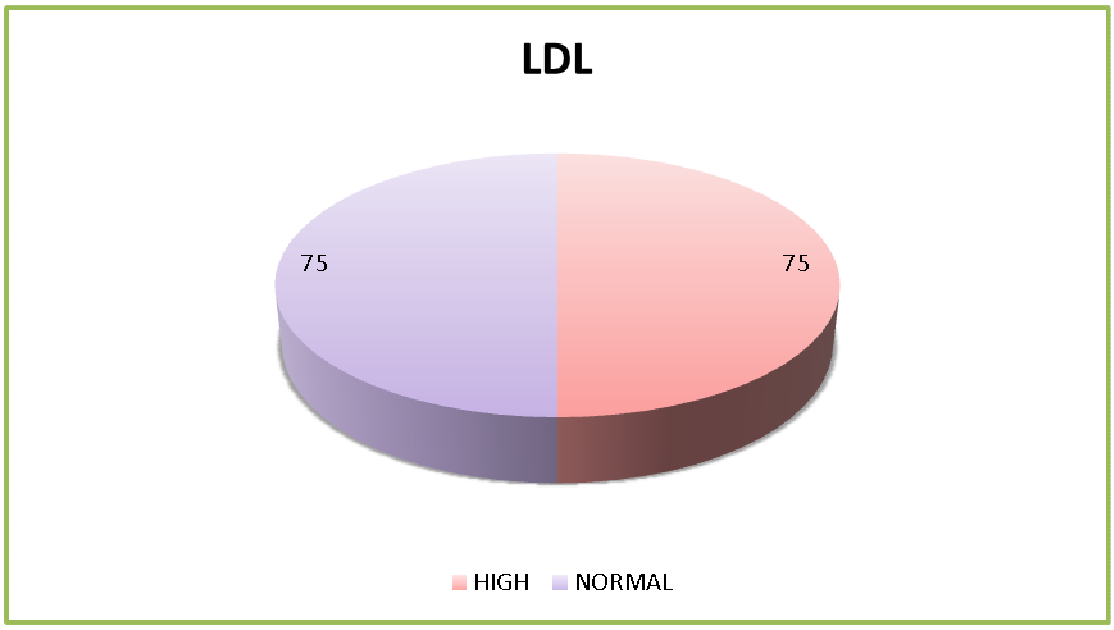
	HDL	
PIH	**MEAN**	**S.D**
PRESENT	56.1	22.33
ABSENT	69.74	42
UNPAIRED T TEST		
P VALUE - 0.012		
SIGNIFICANT		

Mean & S.D. of HDL values is significant for pih (p-value – 0.012)



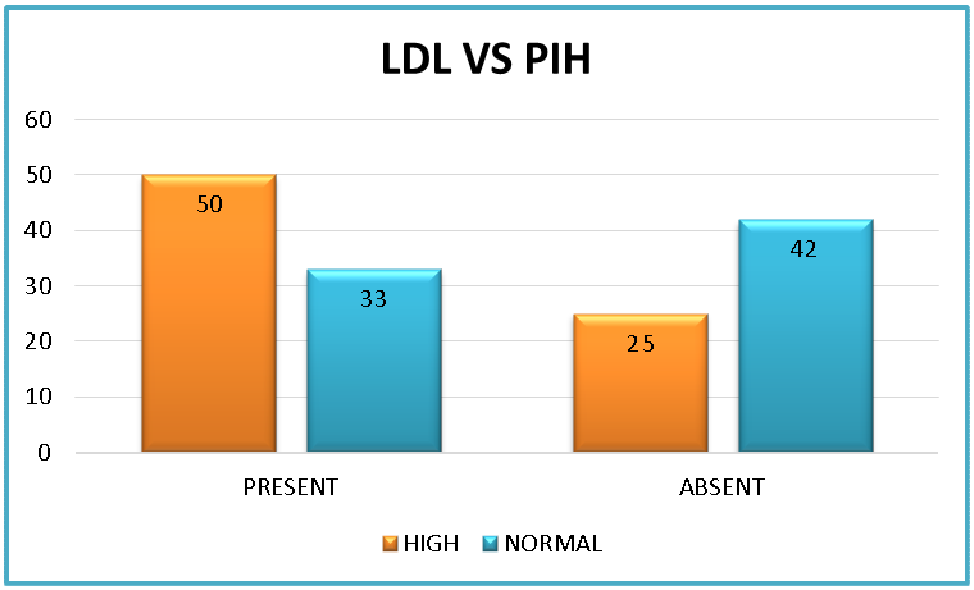
Mean & S.D. of HDL values is significant for pih (p-value – 0.012)

LOW DENSITY LIPOPROTEINS & PIH.



The level of LDL is equally high and normal in 50% of the patients in this study.

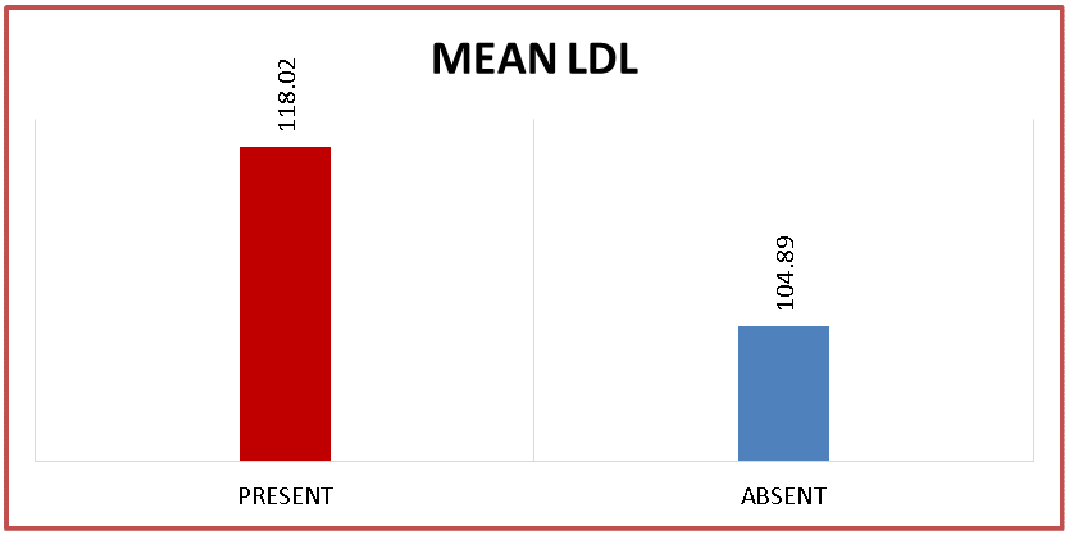
	PIH	
LDL	PRESENT	ABSENT
HIGH	50	25
NORMAL	33	42
P VALUE - 0.005		
SIGNIFICANT		
CHI SQUARE TEST		
ODDS RATIO-2.5		



Of the 150 patients, of the 83 patients developing pih , 50 patients had increased LDL, 33 had normal LDL.The remaining 67 patients had normal LDL who are normotensives.

	LDL	
PIH	**MEAN**	**S.D**
PRESENT	118.02	39.33
ABSENT	104.89	44.62
UNPAIRED T TEST		
P VALUE - 0.05		
SIGNIFICANT		

Hence increase in LDL is significant in developing preeclampsia.

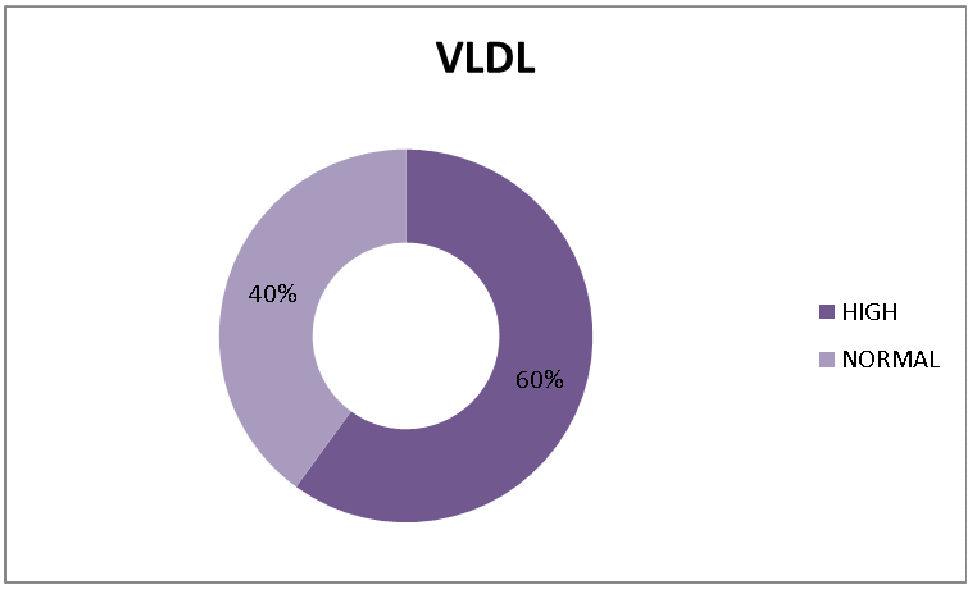


The Mean & S.D. of the LDL is however more than the normal in pih.

VERY LOW-DENSITY LIPOPROTEINS.

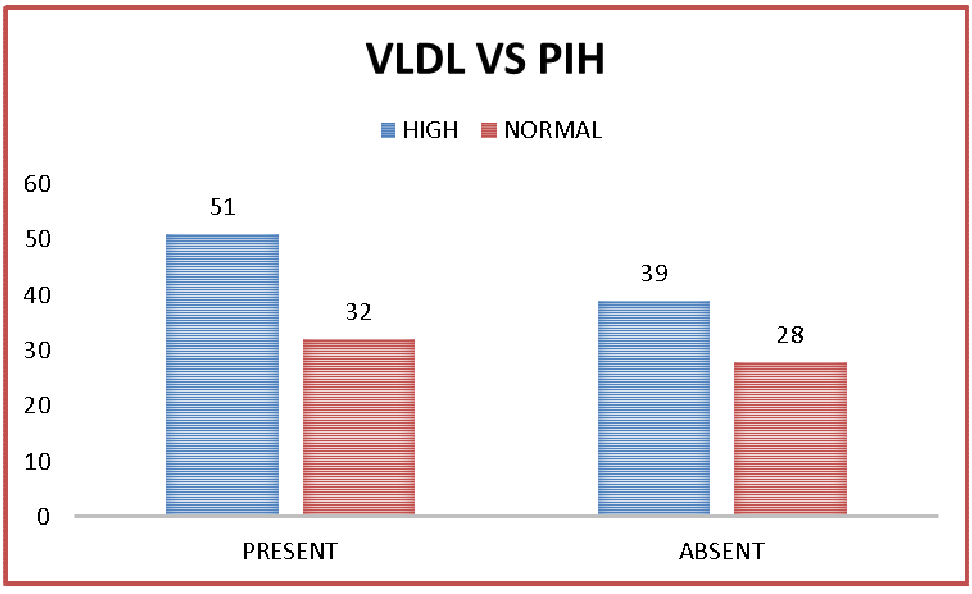
VLDL	NO OF PATIENTS	PERCENTAGE
HIGH	90	60%
NORMAL	60	40%

Generally in this study, the level of VLDL is increased more than 40 in 60%(90 patients of 150).It's normal in 40% (60 patients of 150).



	PIH	
VLDL	**PRESENT**	**ABSENT**
HIGH	51	39
NORMAL	32	28
P VALUE - 0.687		
NON SIGNIFICANT		
CHI SQUARE TEST		

Out of 150 patients, 83 patients developing pih, VLDL is increased in only 51 patients,while the remaining 32 have normal .This proves that VLDL is not significantly raised in all pih patients.(i e. it's non-significant- p-value-0.687) as depicted below.



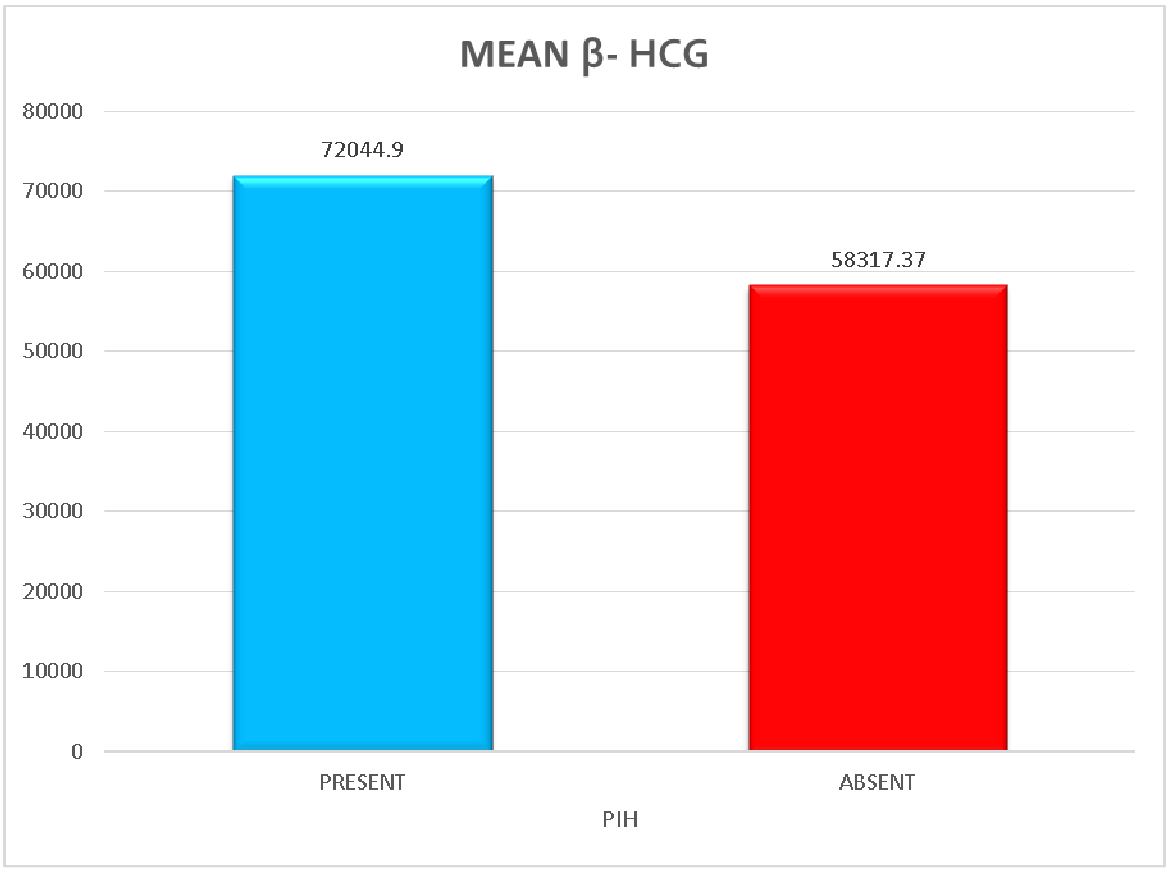
	VLDL	
PIH	**MEAN**	**S.D**
PRESENT	49.82	37.62
ABSENT	47.87	30.7
UNPAIRED T TEST		
P VALUE - 0.734		
NON SIGNIFICANT		

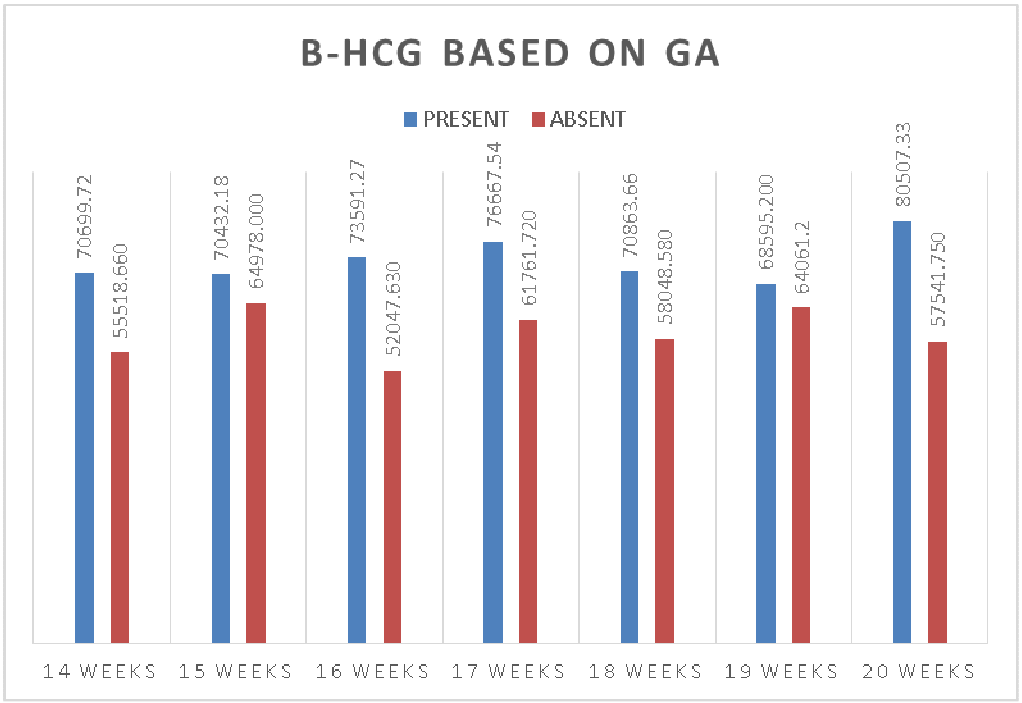
The above table depicts that the mean +- SD of the VLDL is not related to pih by unpaired t test in this study(p-value of 0.734).

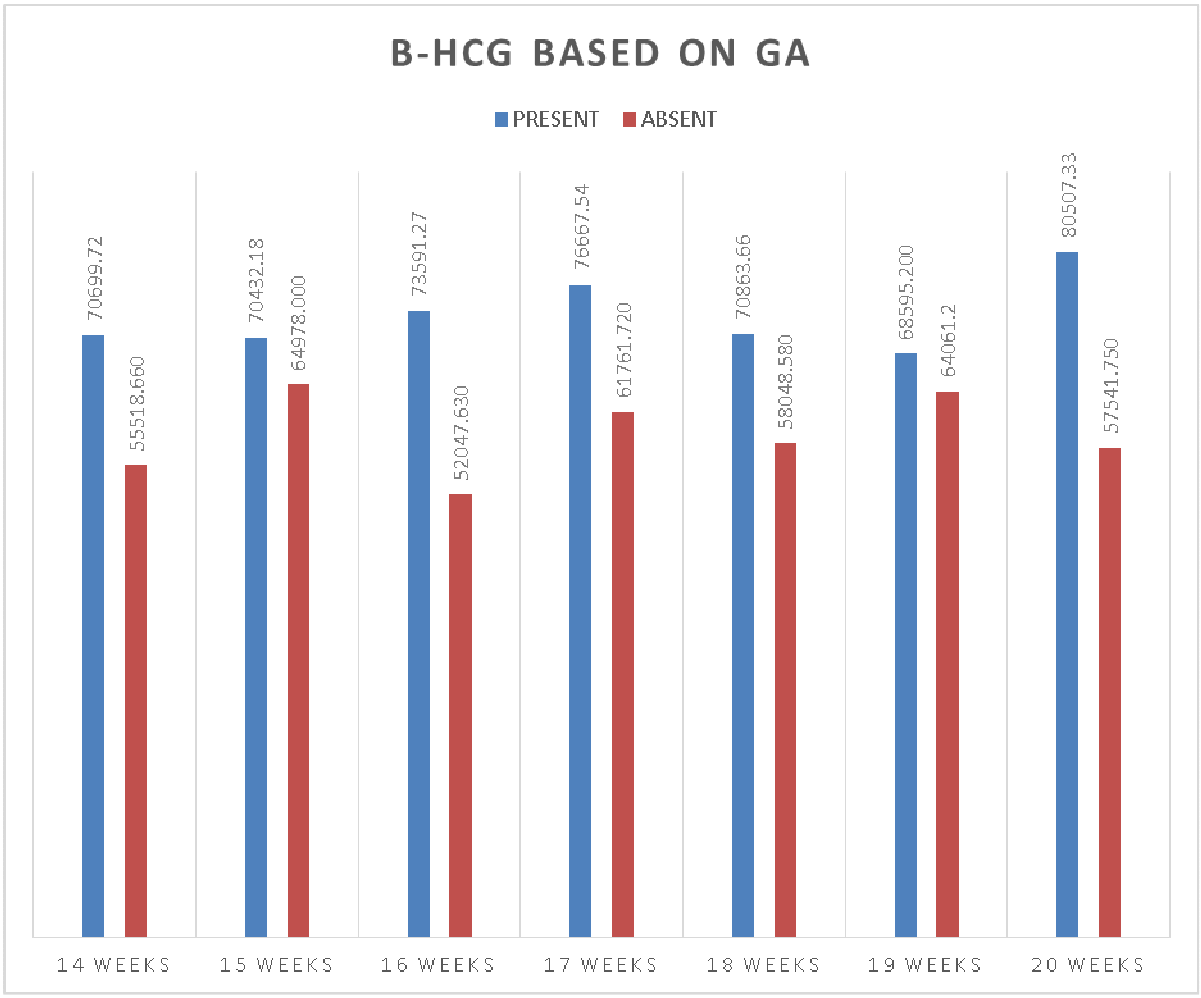
Beta- HCG and it's relation to PIH.

	BETA HCG	
PIH	**MEAN**	**S.D**
PRESENT	72044.9	23649
ABSENT	58317.37	19486
UNPAIRED T TEST		
P VALUE - 0.001		
SIGNIFICANT		

The Mean+- SD of the beta HCG values is significantly more than normal values in this study(14 to20 weeks) by Unpaired t test(p-value is 0.0001).


MEAN β- HCG
80000
70000
60000
50000
40000
30000
20000
10000
0
72044.9
58317.37
PRESENT
ABSENT
PIH


B-HCG BASED ON GA
PRESENT
ABSENT
70699.72
55518.660
70432.18
64978.000
73591.27
52047.630
76667.54
61761.720
70863.66
58048.580
68595.200
64061.2
80507.33
57541.750
14 WEEKS
15 WEEKS
16 WEEKS
17 WEEKS
18 WEEKS
19 WEEKS
20 WEEKS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58048.580
68595.200
64061.2
80507.33
57541.750
14 WEEKS
15 WEEKS
16 WEEKS
17 WEEKS
18 WEEKS
19 WEEKS
20 WEEKS

DISCUSSION

This study focusses on four main hypothesis namely,

1. Placental implantation with abnormal trophoblastic invasion of the uterine vessels.

2. Immunological maladaptive tolerance between maternal, paternal (placental) and fetal tissues.

3. Maternal maladaptation to cardiovascular or inflammatory changes of normal pregnancy.

4.Genetic factors including inherited predisposing genes and epigenetic influences.

PLACENTAL PATHOLOGY:

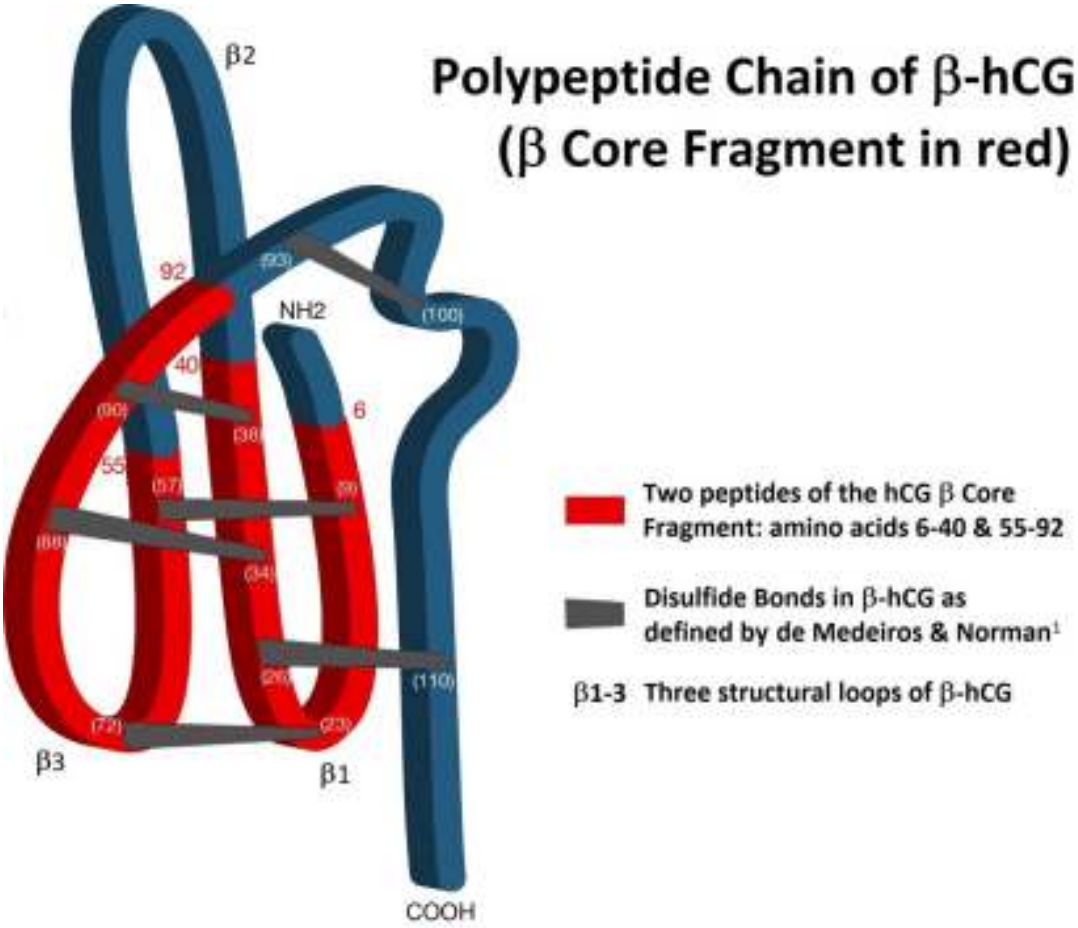
In preeclampsia, there is hyperplacentosis with abnormal placental invasion. Hence, beta- HCG(human chorionic gonadotropin) in pregnancy is increased(due to the failure of the trophoblast invagination and invasion of the spiral arterioles),as a compensatory mechanism to low b-hcg.

BETA-HCG:

It's a glycoprotein composed of 237 aminoacids with a molecular mass of 36.7kDa, approximately 14.5kDa alphaHCG and 22.2kDa

betaHCG. It is a heterodimeric, with alpha subunit identical to that of LH, FSH,TSH and beta subunit that is unique to hCG.

Structure and its function:



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alpha subunit is 92 aminoacids long. beta subunit contains 145 aminoacids(encoded by 6 highly homologous genes arranged in tandem and inverted pairs on chromosome 19q13.3.The two subunits create a small hydrophobic core surrounded by a high surface area-to-volume ratio.The vast majority of the outer aminoacids are hydrophilic.

FUNCTION:

Due to its highly negative charge,hCG may repel the immune cells of the mother protecting the fetus during the first trimester. There is also another hypothesis that hCG may be a placental link for the development of local maternal immunotolerance. Thus, there is a link in the development of peritroohoblastic immune tolerance and may facilitate the trophoblast invasion, which will expedite fetal development in endometrium.

Weeks	**Amount of hCG(mIU/l) Mean Level**	**Amount of hCG(mIU/l) Range of values**
14 weeks	56,600	13,300 – 254,000
15 weeks	50,000	13,300 – 254,000
16 weeks	37,500	13,300 – 254.000
17 weeks	21,500	4,060 – 165,400
18 weeks	24,200	4,060 – 165,400
19 weeks	17,400	4,060 – 165,400
20 weeks	15,700	4,060 – 165,400

LIPID PROFILE IN PREGNANCY:

Studies have shown that the circulating concentrations of triglycerides, low density lipoproteins, high density lipoproteins and total cholesterol increased during pregnancy. This is necessary because of

-the high energy required for the increased cellular proliferation of the maternal uterine enlargement

-Blood volume expansion and fetal implantation as discussed above already.

Pregnancy is a state of increased insulin resistance and insulin secretion and of insulin secretion and of➡reduced hepatic excretion.{Hyperinsulinemia ➡ increased peripheral glucose utilization decline in plasma fasting glucose ➡ increased tissue storage of glycogen➡ increased storage of fats and decreased lipolysis}.

Parameters	Control	I trimester	II trimester	III trimester
TC	137.4+/- 11.4	176.4+/- 18.1	200+/- 13.4	209.3+/- 12.2
HDL-C	40.1+/- 4.4	43.7+/- 3.2	46.0+/- 3.3	54.0+/- 3.8
LDL-C	88.8+/- 13.5	87.3+/- 6.3	127.7+/- 9.9	161.5+/- 12.6
TG	43.7+/- 6.6	138.7+/- 11.1	168.5+/- 10.4	171.7+/- 10.8

Any elevation in the above lipid profile values in second trimester due to altered lipid metabolism is significantly related to preeclampsia in this study as follows.

After getting ORAL & WRITTEN INFORMED CONSENT from all the 150patients ,the study was carried out. Beta hCG is taken from 150 patients in a 12 hour fasting period on all the opd patients, by ELISA test(Enzyme Linked Immuno Sorbent Assay) and they were followed up till term.The samples were collected in association with BIOLINE laboratories and CMCH Biochemistry Lab.

With respect to lipid profile, the samples are collected irrespective of the fasting status. All the lipid parameters were estimated by Enzymatic colour test.

Analysis were made based upon the various characteristics that affect the incidence and frequency of preeclampsia namely, Age, Parity, Pre-pregnancy and Present pregnancy weight, BMI, Gestational Age, Family History, Pedal edema, Blood Pressure(both systole and diastole),Total cholesterol, Triglycerides, HDL, VLDL, LDL and beta hCG.

STATISTICAL ANALYSIS:

It was carried out in terms of mean+- SD for PIH and normotensive cases separately.To analyse the result of our study, we used independent t-test,p-value.Student t-test was employed to compare the mean between PIH and normotensive cases.No binomial logistic regression analysis and Degree of Freedom used.The effects were measured in terms of Odd's Ratio.P-value<0.05 was considered statistically significantMy study is a prospective study.Out of 150 patients, 83 of them developed PIH and the remaining were normotensives.Systolic_blood pressure was elevated in about 57 patients and diastole was elevated in 67 patients.

With respect to AGE,its not that as in other studies,teenage pregnancy doesn't carry a high risk of PIH. Instead ,about 65/83 were between 21 to 30yrs of age. The remaining fall in either extremes(<21 &>30). By Krusker-Wallis test,teenage is not significantly related to pih in this study.By Unpaired t-test, the mean+_SD of the age is not significantly related to pih(p-value-0.832).

In reference to WEIGHT,there is a significant rise in weight from the pre-pregnancy range to present pregnancy according to gestational age irrespective of the incidence of pih. However,there is EQUAL rise in weight among both normotensives and PIH patients in this study.This is explained by Unpaired t-test where the mean+_SD of the pre-pregnancy

& present pregnancy weight is not signifcanly related to PIH.(p-value-0.995 & 0.977).

While quoting about FAMILY HISTORY,not all those who develop pih had positive family history. Only 38 of 83 patients had positive family history.

PARITY:

Generally, it is seen that PIH are more common in primigravida. But in this study,66 were primi, 60 belonged to 2^{nd} gravida & 24 were > 2^{nd} gravida. By Chi-square test,the incidence of PIH is equivocal, i.e.(out of 83 pateints, incidence -primi= 2^{nd} gravida with p-value-0.441- not significantly related to p ih).

GESTATIONAL AGE:

In my study, the gestational age where the incidence of preeclampsia can be screened or predicted more positive is at 16 weeks of gestation.The Mean +_ SD of the gestational age is significantly related to PIH(p-value-o.037) by Unpaired t-test.

BODY MASS INDEX:

Since the pre-pregnancy and present pregnancy weight are almost equal to that of normal pregnancy (i.e. same rise is observed in pih patients),BMI is also not significantly related to pih by unpaired t-test(p-value-0.105).

PEDAL EDEMA:

With regard to pedal edema,out of the 150 sample , 66 had pedal edema(grade-2),of which 49 developed pih in the subsequent antenatal visits till term. Hence, by Chi-square test,it is significantly related to preeclampsia(p-value-0.002) with Odd's ratio(4.2).

TOTAL CHOLESTEROL:

In my study, 52 out of 155 had total cholesterol >200 mg/dl,out of which,37/52 developed pih ,the remaining 15 had normal cholesterol values with pih.The 98/155 had normal cholesterol levels.The Mean+-SD of the TC was significantly related to PIH(p-value – 0.013) by Unpaired t-test.{i.e.229.33-pih & 203.51-normotensives}.

TRIGLYCERIDES:

Out of 150 patients,only 8 out of 142 patients had increased triglyceride levels.The Mean+- SD of the triglcerides is not significantly related to PIH(p-value-0.585).

HIGH DENSITY LIPOPROTEINS:

HDL is normally not elevated in pih.However decreased HDL leads to rise in pih.In our study, out of 150 patients, 58 had low HDL<40 mg/dl,the remaining 92 had normal levels.Out of 58 patients,44 developed pih.Hence by Chi-square test,HDL is not significantly related to pih(p-value-0.316).However,by Unpaired t- test, the Mean+- SD of HDL is significantly related to PIH(p-value-0.012).

LOW DENSITY LIPOPROTEINS:

In our study, out o f 150, 75 patients had high LDL levels>150, the remaining 75 had normal LDL levels.Among 75 patients, who were having increased LDL levels,only 55 out of 83(who developed pih) had high LDL levels and the 30 had normal LDL levels. By Chi-square test, the levels of LDL is significantly related to pih(p-value-0.005 and Odd.s ratio- 2.5).

VERY LOW DENSITY LIPOPROTEINS:

Out of 150 patients,VLDL is increased in 90/150 patients and 60 had normal levels.Among 90,51 subsequently developed pih and 39 were normotensives.Og the 60 patients,32 manifested wih pih symptoms even with normal VLDL levels and 28 wer normotensives.Hence, by Chi-square test, VLDL is not significantly related to pih(p-value – 0.687).By Unpaired t-test,the Mean +_SD is not significantly related to pih(p-value-0.734).

Mossink et al and Pouta et al concluded that "rise in b-hcg was not observed in gestational hypertension cases but only in severe preeclampsia",

Lorentzen et al concluded that "serum- free fatty acids and triglycerides were increased before 20 weeks of gestation and later they developed preeclampsia."

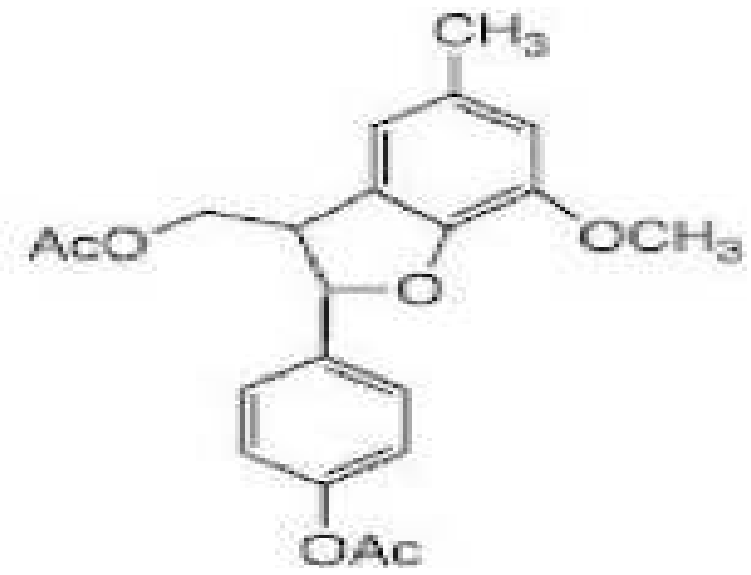
Cekemen et al showed that " plasma triglycerides &LDL were higher in preeclampsia,whereas HDL were lower in preeclamptic patients".

De et al concluded that " triglycerides and VLDL were raised and HDL levels decreases in preeclamptic patients".

Vidyabati et al showed that "total cholesterol,VLDL,LDL were significantly raised in preeclampsia patients".

Beta hCG:

The Mean+_SD of the beta hCG values is significantly related to pih between 14 to 16 weeks of gestation with more predilection to 16 weeks.{pih -72,044.9+_ 23,649 and normotensives – 58,317.37+_ 19,486}.



HCG Molecular Structure

CONCLUSION

From this study, the following conclusions have been arrived which are different from the previous studies that need large sample size to bring about these parameters as predictors of pregnancy induced hypertension.

Among the various factors as mentioned above(which influence the incidence of pregnancy induced hypertension), five are only significantly related to PIH in my study.

1.Of the 150 patients, 66 had pedal edema & nearly 49 developed pih(p-0.004).

2.The Mean+_SD of the total cholesterol levels were significantly related to pih-{by Unpaired t-test, the p-value-0.013,with PIH-229.33;Normotensives-203.51}.

3.By Unpaired t- test, the Mean+_SD of the HDL was indirectly related to PIH(p-value- 0.0012).

4.The Mean+- SD of the LDL levels were significantly related to PIH by Unpaired t-test{p-0.05}.

5.The Mean +_ SD of the beta hCG were directly related to PIH according to gestational age included in this study {i.e.72,044.9+_23,649 in PIH and 58,317.37+_19,486 in Normotensives}.

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CONSENT FORM

NAME :

AGE :

SEX :

ADDRESS :

I, DR.S.NITHYA, doing post graduation in the department of obstetrics and gynaecology in COIMBATORE MEDICAL COLLEGE HOSPITAL, is undergoing a research study on the antenatal patients upon the topic, "SERUM BETA HCG AND LIPID PROFILE AS THE EARLY PREDICTORS OF PREGNANCY INDUCED HYPERTENSION BETWEEN 14 TO 20 WEEKS OF GESTATION". I have cleared all my doubts regarding the topic from the guide of my department. I have thoroughly known the essence of the topic. I hereby declare that I'm participating with the full involvement in the study. I will protect all the information obtained in the study. It is known the essence of the topic. I hereby declare that I'm participating with the full involvement in the study. I will protect all the information obtained in the study. It is known to me that I have the right to withdraw myself from my study at any point of time.

PLACE:

DATE :

SIGNATURE

ஒப்புதல் படிவம்

பெயர் :

வயது :

பாலினம் :

முகவரி :

கோவைஅரசுமருத்துவக் கல்லூரியில் மகப்பேறுபிரிவில் பட்டமேற்படிப்பு பயிலும் மாணவிமரு.ச.நித்யாஅவர்கள் மேற்கொள்ளும் ஆய்வில் மகப்பேறுபெண்களுக்குரத்தஅழுத்தம் அதிகமாகும் திறன் 14 வாரம் முதல் 20 வாரம் வரைஅதிகமாக இருப்பதைBETA HCG(பீடாஹெச், சி.ஜி) மற்றும் LIPID PROFILE (கொழுப்பு சத்து) என்னும் இரு ரத்தப் பரிசோதனைகள் மூலம் முன்னதாகவேஅறியலாம் என்பதைபற்றியஅனைத்துவிளக்கங்களையும் கேட்டுக் கொண்டுஎனதுசந்தேகங்களைதெளிவுபடுத்திக் கொண்டேன் என்பதைத் தெரிவித்துக் கொள்கிறேன். நான் இந்தஆய்வி முழு சம்மதத்துடன் கலந்துகொள்ளஒப்புதல் அளிக்கிறேன். இந்தஆய்வில் என்னைப் பற்றிஅனைத்துவிவரங்கள் பாதுகாக்கப்படுவதுடன் இதன் முடிவுகள் ஆய்வில் வெளியிடப்படுவதில் ஆட்சேபனை இல்லைஎன்பதைதெரிவித்துக் கொள்கிறேன். எந்தநேரத்திலும் இந்தஆய்வில் இருந்துவிலகிக் கொள்ளஎனக்குஉரிமைஉண்டுஎன்பதைஅறிவேன்.

இடம் :

தேதி:

கையொப்பம் / ரேகை

ANNEXURE

PROFORMA

NAME :

AGE :

OCCUPATION :

HOSPITAL OP NO. :

ADDRESS :

CHIEF COMPLAINTS :

MENSTRUAL HISTORY :

Last menstrual period -LMP

OBSTETRIC HISTORY :

Parity and history of preeclampsia in the previous pregnancy.

PAST HISTORY :

FAMILY HISTORY :

History of pregnancy induced hypertension in the family members &

History of hypertension in the family members.

GENERAL EXAMINATION :

HEIGHT :

PRE-PREGNANCY WEIGHT :

PRESENT PREGNANCY WEIGHT:

PEDAL EDEMA :

BODY MASS INDEX :

USG examination :

Preferably dating scan or early second trimester scan to calculate the gestational age,in case of irregular cycles(between 14 to 20 weeks of gestation).

INVESTIGATIONS :

Serum beta hCG :

Serum Lipid Profile :

TOTAL CHOLESTEROL :

LDL :

VLDL :

HDL :

ABBREVIATIONS

SD	- Standard deviation
hCG	- Human Chorionic Gonadotropin
HDL	-High Density Lipoproteins.
LDL	-Low Density Lipoproteins.
VLDL	-Very Low Density Lipoproteins.
TC	- Total Choloesterol
TGL	- Triglycerides.
PE	- Pedal Edema
BMI	-Body Mass Index.

KEY TO MASTER CHART.

GEST. – Gestational Age.

PRE WT - Pre-pregnancy weight

PIH -Pregnancy induced hypertension.

Code no. 1- PIH present

2- PIH absent.

PE - Pedal edema.

BMI -Body Mass Index.

SBP - Systolic Blood pressure.

DBP - Diastolic Blood pressure.

TC - Total Cholesterol

TG -Triglycerides.

HDL - high density lipoproteins.

LDL - low density lipoproteins.

FH

NAME	AGE	HEIGHT	PRE WT	FH	PARITY	GEST.	NOW WT	BMI	SBP	DBP	PIH	PE	BETA HCG	TC	TG	VLDL	HDL	LDL
Rajmathi	19	142	42	no	primi	19	46	22.8	120	90	1	no	51262	164	221	23.5	48	145
Anitha	19	160	60	yes	primi	14	64	25	140	90	1	yes	57613	210	154	30.5	42	150
Saranya	25	152	51	no	G2A1	15	55	23.8	130	90	1	no	61650	250	151	30.5	46	125
Sajitha	24	153	53	no	G2A1	15	56	23.9	130	80	2	no	52300	245	142	41.8	56	115
Sarasal	25	157	57	yes	G2P1L1	16	61	24.7	140	70	1	yes	62650	254	161	52.8	62	140
Priyanka	27	154	54	no	G2P1L1	18	57	24	130	80	2	no	47512	210	150	45.8	35.3	95
Paseena	21	154	54	yes	primi	17	58	23.7	130	80	2	yes	39451	285	120	44	31	80
Sathybama	21	145	45	no	primi	17	48	22.8	120	80	2	no	58615	175	135	41.5	32.4	82.1
Devika	21	144	44	no	G2A1	14	47	22.7	100	60	2	no	48530	210	125	31.5	33.5	89
Dhivya	22	148	48	no	G2P1L0	20	52	23.7	130	60	2	no	57252	174	135	25.1	86	166
Dhevaki	20	147	46	no	primi	20	50	23.1	130	60	2	no	47125	211	150	24	25.1	174
Sathya	24	149	49	no	G2A1	18	53	23.9	130	80	2	yes	59123	174	135	25.2	38	62
Surya	26	148	49	yes	G2P1L1	18	52	23.4	130	90	1	no	61150	164	140	27.8	45	101
Girija	26	154	54	yes	G2P1L0	16	57	24	140	100	1	yes	67512	195	145	31.2	41	154
Gomathi	24	160	60	no	G2A1	16	63	24.6	140	100	1	no	65110	270	250	45	65	162
Dhayalini	28	162	62	no	G3P2L2	16	65	24.8	120	70	2	no	41000	154	115	50.1	31.5	61.5
Sumathy	31	164	63	no	G2P1L1	15	66	24.5	110	70	2	yes	75123	170	110	51.1	32.3	62
Sajana	34	161	60	yes	G3P2L2	14	64	24.7	130	80	2	no	63154	454	290	57.5	58	124
Sandy	20	154	54	yes	G2A1	15	58	24	140	80	1	yes	72524	212	148	40.8	45	128
Saratha	19	165	65	yes	primi	15	68	25	140	90	1	yes	71650	250	152	31.5	48	135
Tamil	24	162	62	yes	G2P1L0	16	65	24.8	150	90	1	yes	57650	201	156	31.2	47.5	100
Roja	35	155	55	no	G3P2L2	18	57	23.7	130	80	2	yes	59150	225	165	30.5	46.2	98
Rabiya	30	152	52	no	G2P1L1	18	55	23.8	120	80	2	yes	58154	215	171	29.5	43.1	76
Rasathi	27	149	49	no	primi	16	54	24.3	120	60	2	no	61125	215	165.5	28.5	42.5	130
Sanshya	25	148	48	no	primi	17	52	23.7	130	60	2	no	58123	175	145.5	45.2	81.7	146
Santhana	24	150	50	no	primi	18	56	24.9	150	60	1	no	84151	300	175.5	36.5	65.6	179
Shanthi	21	152	52	no	G2A1	14	55	23.8	140	90	1	no	66523	190	185.2	31.2	75.1	149.7
Christine	22	158	58	yes	G2A1	14	63	25.2	110	70	2	no	63151	165	189	35.8	89	150
Chinathai	21	157	57	no	primi	14	61	24.7	120	80	2	no	40125	185	191.1	32.2	84	89
Devarani	20	163	63	no	primi	15	66	24.8	140	100	1	yes	72150	256	180.5	32.1	83.5	134
Eshwari	26	165	65	no	primi	15	68	25	130	80	2	no	51140	110	140	42.2	31.5	99
Firdouse	26	158	58	no	G2P1L1	16	62	24.8	130	80	2	no	31520	161	270	171.5	41.1	61.5
Fathima	25	154	54	yes	G2P1L1	17	60	25.3	140	70	1	yes	48868	154	135.5	45.1	50	136
Hindumi	24	152	52	no	G2P1L1	18	55	23.8	160	70	1	no	59320	182	125	46.1	28	154
Anuradha	19	154	54	yes	primi	14	57	24	120	80	2	yes	100000	155	147	40.5	43	98
Ambhika	25	161	61	no	G2P1L1	15	64	24.7	120	100	1	yes	98567	276	155	41.8	40	110
Bharkavi	24	161	60	no	G2A1	15	64	24.5	140	90	1	yes	57165	247	165.5	71.8	45	125
Pappathi	24	151	50	no	G2P1L1	16	53	23.2	140	80	1	yes	58110	248	168.8	41.1	46	93
Viji	25	147	47	no	G3P2L1	15	50	23.1	140	80	1	yes	61501	200	275	41.3	63	82
Parvadhi	26	148	49	yes	primi	14	52	23.7	160	100	1	yes	71150	165	135	40.2	42	135.4
Rajammal	23	150	50	no	primi	14	53	23.6	170	90	1	no	60050	275	133	44.5	78	80
Raji	28	155	55	no	G3P1L2	16	58	24.1	140	80	1	no	58125	154	95	43.2	45	75
Anaporni	21	167	67	no	primi	18	70	25.1	130	80	2	no	61458	150	92	31.7	80	195
Bhuvani	21	153	54	no	primi	18	57	24.3	130	80	2	yes	57500	215	185	27.1	85	90
Preetha	20	157	57	no	primi	19	61	24	130	60	2	no	65160	125	75	27.5	87	85
Mangala	20	154	54	no	G2A1	18	58	24	120	60	2	no	102453	226	165.7	28.5	42.5	185
Maral	19	149	49	no	G2A1	20	53	24.3	110	80	2	no	101100	226.6	165.8	31.5	44.5	95.5
Ponni	20	152	52	no	primi	18	55	23.2	140	100	1	yes	58176	175	185.7	32	52.2	137.9
Malar	21	157	57	no	primi	18	60	24.7	140	90	1	no	55110	165.5	195.4	327	52.1	190.6
Rani	21	150	50	no	G2A1	16	55	24	140	80	1	no	56430	254	169.2	41.5	45	101.9
Lakshmi	23	168	69	no	G2P1L1	16	74	26.2	120	90	1	no	54135	145.2	171.4	50.4	26.5	125

Mathi	24	165	65	yes	primi	14	68	25	120	90	1	no	62350	174	156	50	65	125
Priyanka	21	154	54	yes	G2A1	16	57	24	110	80	2	no	37512	250	170	31.5	60	100
Bharathi	23	174	74	no	G2A1	17	77	25.4	130	90	1	yes	54860	214.1	180	25.5	43	115
Mehboob	22	143	43	no	G2A1	15	47	22.5	140	90	1	no	76523	261	145.1	45	75.2	124
Chithra	24	148	49	no	primi	15	52	23.7	140	90	1	yes	41150	261.5	140.3	42.5	71.2	175
Sindhu	28	157	58	yes	G3P2L2	18	62	25.2	130	80	2	no	60150	211	125.3	41.5	78.4	125
Durga	27	139	39	yes	G3P2L2	19	42	21.7	120	80	2	no	65213	140	100	31.5	85	135
Shantha	25	144	45	yes	GP1L1	16	48	23.1	110	90	1	yes	51110	145	177.5	25.4	42.4	140
Pooja	21	156	56	no	primi	20	59	23.8	140	100	1	yes	85160	175.1	165.4	27.5	41.2	150
Brindha	21	162	62	yes	primi	20	66	25.1	140	80	1	no	95160	213.5	172.1	28.5	32.2	160
Sathya	19	170	69	yes	G2A1	16	72	24.9	140	90	1	no	81140	264	195.5	81.5	35.5	115
Sundhari	18	148	48	no	primi	14	54	24.7	150	110	1	yes	92140	222.7	181.4	42.5	32.5	125.9
Selvi	22	139	40	no	primi	16	44	22.3	140	90	1	no	55620	195	172.5	51.6	31.5	135
Thamarai	23	145	45	no	G2P1L1	17	49	22.8	130	70	2	no	31526	185.4	140	74	27.5	109
Deivanai	24	149	50	yes	primi	17	55	24.6	130	90	1	yes	45160	175.4	113.5	85.5	24.5	95
Sindhi	24	174	74	no	G2A1	19	78	25.8	130	90	1	yes	41150	165.4	127.5	82.6	21.5	98
Meena	23	161	61	no	G2P1L1	15	64	24.7	120	90	1	no	74150	181.3	100.5	51.2	20.5	97
Vaidhegi	27	154	54	no	G3P1L1A1	14	57	24	120	80	2	no	30120	150	140	35	45	124
Vasanthi	21	158	58	no	primi	16	61	25.2	120	80	2	no	38621	149	130	40	43	55
Payal	21	152	53	no	primi	18	56	23.8	130	80	2	no	59160	165	185	45	41	74
Basanthi	25	148	48	no	G3P1L1A1	20	52	24.7	130	80	2	no	75421	210	170	42	63.6	171
Aarthi	24	162	62	no	G2A1	17	65	25.1	150	90	1	yes	71750	225	185	93	49	101
Amudha	24	141	41	no	G2P1L1	16	45	22.1	110	80	2	yes	68150	240	190	103	51	158
Pallavi	23	145	45	yes	primi	17	48	23.1	120	70	2	no	65123	200	195	115	56	115
Bairavi	22	139	39	yes	primi	16	42	21.7	120	60	2	yes	41150	154	200	160	65	145
Celine	21	165	65	no	primi	17	68	25	110	60	2	no	59123	155	135	98	96	175
Darshini	21	174	74	no	primi	15	77	25.4	110	70	2	no	72154	160	124	25	24	85
Jamuna	26	165	65	no	G2P1L1	15	69	25	110	80	2	no	100010	110	95	45	31	135
Ishwarya	25	145	46	yes	G2P1L1	16	50	23.2	140	90	1	yes	88150	210	175	44	75	114
Lavanya	25	158	58	yes	G3P1L1A1	17	62	25.2	150	100	1	no	151000	240	180	65	40	110
Lalitha	24	149	49	no	G2P1L1	17	54	23.1	170	90	1	yes	100000	220	150	74	84	141
Gayathri	27	140	40	no	G3P2L2	16	44	22.3	140	100	1	yes	95150	270	210	84	74	145
Oorvasi	26	165	65	no	primi	17	68	25	150	70	1	no	84120	300	165	91	45	175
Menaka	21	146	46	no	primi	15	50	23.2	120	70	2	no	69213	310	185	90	144	200
Fahsina	22	154	54	yes	primi	14	57	24	130	90	1	yes	95150	215	190	100	81	150
Pavithra	21	159	59	no	primi	16	63	24.8	120	90	1	no	59150	250	179	66	45	150
Banupriya	22	147	47	no	primi	14	50	23.1	130	90	1	no	61150	210	175	36	95	145
Menaka	24	146	46	no	G2P1L1	17	50	23.2	130	80	2	no	74581	255	176	56	92	211
Valar	37	143	43	yes	primi	15	47	23	140	80	1	yes	84110	310	167.4	71	85	237
Nithya	30	148	48	yes	G3P2L2	16	51	28.3	150	80	1	yes	184000	440	200	81	42	155
Sindhu	21	154	54	no	G2A1	20	60	25.3	110	80	2	no	50000	200	176	25	48	91
Sharvanai	19	165	65	yes	primi	19	68	25	110	80	2	yes	75412	149	189	38	57	95
Thilagam	23	147	47	no	G2P1L1	14	51	23.6	120	80	2	no	65215	165	198	98	25	80
Sairani	18	174	74	no	primi	16	77	25.4	130	80	2	no	65150	268	155	49	35	62
Geetha	23	135	35	yes	primi	15	39	21.4	130	80	2	yes	65210	125	175	87	38	110
Bhavani	19	145	45	yes	G2A1	14	52	29.1	140	90	1	yes	92000	215	165	80	49	148
Mallika	27	148	48	no	G3P2L1	17	51	23.3	120	90	1	no	52102	210	174	65	41	75
Latha	26	152	52	no	G2P1L1	14	55	23.8	110	90	1	no	70010	150	132	40	28	65

Daisy	25	164	64	yes	G2P1L1	16	68	25.3	110	70	2	no	46150	145	135	40	28	65
Sudar	24	170	70	no	G2A1	18	74	25.4	110	60	2	no	61320	168	127	41	56	130
Nancy	25	158	58	no	G2P1L1	14	61	24.4	130	90	1	no	57140	175	150	25.5	31	95
Evangel	24	160	60	no	primi	18	64	24.6	120	70	2	no	60124	162	154	45	28	97
Maha	26	148	48	no	G3P1L1A1	15	51	23.3	130	60	2	no	24510	200	174	40	25	85
Sabrina	28	147	47	yes	G3P2L2	16	51	23.6	110	80	2	no	71245	250	160	65	47	165
Poongodi	20	160	60	yes	primi	18	65	25.7	140	90	1	yes	98176	226	148	74	44	115
Panimalar	20	144	44	yes	G2A1	14	47	22.7	140	90	1	yes	72000	150	174	71	40	74
Bindhu	19	167	67	yes	primi	16	70	25.1	150	90	1	yes	56451	145	165	54	37	78
Aanandhi	18	169	69	no	primi	17	73	25.2	160	110	1	yes	77154	265	124	36	65	125
Aarthi	27	165	65	no	G3P2L1	18	69	24.2	140	100	1	yes	89176	400	130	65	75	134
Mohana	24	146	46	yes	primi	16	51	20	140	100	1	yes	91150	510	101	74	85	155
Beevi	21	148	48	no	primi	15	52	24.4	140	100	1	yes	98064	250	151	25	80	198
Gokila	22	160	60	no	primi	17	64	24.7	140	90	1	yes	68908	210	110	81	74	91
Harini	23	163	63	yes	G2P1L1	17	66	24.8	120	80	2	yes	76152	350	115	74	65	158
Shankari	23	167	67	no	G2P1L1	15	70	25.1	140	90	1	no	50152	175	121	31	50	144
Thangam	24	170	70	yes	primi	15	73	25.3	130	80	2	no	75142	214	176	45	78	189
Jothi	21	174	74	yes	primi	16	73	26.1	130	80	2	yes	75123	235	185	74	84	145
Vani	36	169	69	yes	G3P2L2	17	74	26.2	140	100	1	yes	89421	265	145	81	42	190
Rathinam	19	148	48	no	primi	14	52	24.4	130	100	1	yes	67189	250	134	56	60	137.4
Jeya	25	152	52	yes	G2P1L1	16	55	23.8	120	90	1	no	83000	286	129	45.6	67.5	115
Sonia	21	147	47	no	primi	18	51	23.6	140	90	1	no	65341	200	143	34	41	128.8
Shakthi	26	163	63	no	G2P1L1	19	67	24.9	130	90	1	yes	145000	260.7	190	57	89.4	136
Jothi	22	148	48	yes	primi	15	51	22.8	140	90	1	yes	54253	320	156	3l.5	59	55
Mehraj	**22**	152	51	yes	primi	20	54	23.4	110	70	2	yes	38512	215	185	24.3	178	58
Janaki	33	157	58	no	G2P1L1	18	60	24.3	140	90	1	yes	69243	205	172	21.2	70	64
Gayathri	26	154	54	yes	G2A1	14	58	24.5	150	100	1	yes	95713	265	175	21.4	82	50
Vidhya	24	153	52	no	G2P1L1	15	55	23.5	140	90	1	no	65893	185	140	22	98	64
Fathima	28	152	52	nil	G3P2L2	16	55	23.4	120	70	2	yes	9844	175	140	21	185	62
Shanthi	39	155	52	nil	primi	18	58	23.3	120	80	2	no	10479	165	135	21.5	210	65
Vijaya	23	160	60	yes	G3A2	20	64	25	150	90	1	no	61202	211	115	23.1	127	61.5
Sangeetha	30	148	48	no	primi	20	55	25.1	130	80	2	no	58412	291	446	89.3	92	54.8
Hemalatha	27	165	65	no	primi	14	70	25.7	120	70	2	no	34123	215	160	22.4	110	43.2
Sasikala	25	160	60	no	G2A1	14	64	25	120	90	1	yes	57265	220	155	20.9	150	52.6
Selvarani	25	162	62	no	G2P1L1	14	65	24.8	120	90	1	yes	78140	195	101	20.3	45	31.4
Chitrakala	31	154	54	no	primi	19	58	24.5	130	90	1	yes	36521	270	184	30.5	65	58
Sneha	30	158	58	yes	G3P2L2	18	62	24.8	130	90	1	no	98156	240	151	43.2	59	89
Manju	21	161	60	yes	primi	17	65	23.5	130	80	2	no	85963	250	160	65.4	112	68
Lakshmi	25	145	42	yes	G2P1L1	14	47	21.9	110	90	1	no	57012	254	158	21.4	41	49
Nithya	21	151	50	no	primi	16	54	23.7	110	80	2	no	57600	276	175	25.5	105	45
Menaka	23	152	~~52~~	no	primi	17	55	23.8	120	80	2	yes	89199	212	184	26.7	112	49
Aishwarya	28	154	55	no	G3A2	19	59	24.9	130	90	1	no	46412	217	157	23.2	58	68.6
Ranjini	24	155	55	yes	primi	15	60	25	140	90	1	no	87413	285	151	22.4	65	64
Maghi	29	158	58	no	primi	18	62	24.8	150	80	1	yes	48238	232	140	21.3	56	68
Jeeva	30	142	40	no	G6P3L0A4	18	44	21.8	160	80	1	yes	64127	247	140	22.5	51	78
Rasheetha	21	141	41	yes	primi	14	45	22.6	140	80	1	yes	60000	251	138	21.5	54	112
Fahseena	23	145	44	no	G3P1L1A1	17	50	23.8	130	80	2	no	41523	185	141	23.6	124	61
Cathrine	24	158	59	no	G2P1L1	20	64	25.6	120	80	2	no	32512	194	152	21.2	126	52
Rajlakshmi24	24	150	52	yes	primi	14	55	24.4	130	80	2	yes	55250	185	141	21.9	126	54
Pavithra	23	164	65	no	G2A1	16	69	25.7	130	80	2	no	57261	154	235	23.2	131	60
Padma	27	158	57	no	G3P2L2	16	62	24.8	120	80	2	no	79262	435	154	28.2	145	58